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Some Observations on Milk Fever*

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Historical

Milk fever has been recognised and recorded in Britain since the early part of the nineteenth century, and over 70 years ago Professor Walley, who was then Principal of Dick's College, read an essay on the subject to the Scottish Metropolitan Veterinary Association[16] in which he held "that the disease was not due to any disorder of the blood, as that condition is generally understood—as proved by the fact that no evidence of such disorder was ever discernible in *post-mortem* examination—but that it had its origin, primarily, in a paralysed condition of the sympathetic nerve which again led to congestion of the minute capillaries of the spine and brain, leading to progressive paralysis from behind forward." And he went on to state that he "was inclined to attribute the neuro-spinal innervation to mechanical influences exercised on the sacro-lumbar plexus, or to mechanical pressure exerted on the abdominal sympathetic during the act of parturition itself." And with these influences he "associated barometrical and thermometrical influences."

Well, fortunately, I do not need to analyse this theory in detail because within a few years, Professor Walley had abandoned it, and in 1893 we find him[16]

". . . as fully convinced as I can well be in the absence of demonstrable proof that the malady is of toxic origin but that the toxic element is rather of the nature of a leucomaine than of a ptomaine. All ideas as to the poisonous principle having a putrefactive origin are negatived by the clinical characters and course of the malady, but these are in exact accord with the view that it is due to a toxine originating in excessive cytolysis or histolysis—processes which go on at an extremely rapid rate at the preparturient and parturient periods."

I draw attention to this theory (originally attributable, I believe, to Barlow) because quaint as it may seem to us nowadays, it was this, in one or other of its many various forms, which was to be responsible for the more spectacular advances which history was later to record. Thus, as you know, only four years after Walley's statement, Schmidt, acting on the theory that there was a toxin being produced in the udder, injected an antiseptic in the form of a relatively small amount of an aqueous solution of potassium iodide with results that were surprisingly successful. And more than a quarter of a century later it was to lead Dryerre and Greig[6] to evolve in this college a theory which was to be the first step to fame if not to fortune. For they state "we have been impressed by the idea that milk fever arises from the action of a toxin which may be of endogenous or exogenous origin, although not necessarily a bacterial product; as has been indicated, there are difficulties in reconciling the intoxication hypothesis with the cure effected by intra-mammary insufflation, and in seeking for an explanation we have considered that such would probably be found in the field of endocrinology."

Accordingly, stimulated by the advances which had been made in the preceding decade in that field, they enunciated their classical theory which Greig[11] was later to designate "Auto-intoxication consequent upon endocrine disorder" in the following terms:

"We suggest that the animal is suffering primarily from a parathyroid deficiency. The parathyroids cannot cope with the increased metabolism incidental to pregnancy and parturition. Faulty metabolism sets in, and an accumulation of protein toxins (*e.g.*, guanidin) gathers in the body; the formation of guanidin in the bowel and its absorption are facilitated by intestinal stasis and its associated intestinal derangements. There is also a diminution in available calcium. The tissues are on the point of reacting to this condition when relatively slight excitement, by releasing a certain quantity of adrenaline, sensitises the tissues and precipitates the crisis. A form of tetany results. Insufflation of the udder, by initiating powerful afferent stimuli, reflexly causes an excessive secretion of adrenaline and possibly prevents further calcium loss. The adrenaline now exerts its power of hastening oxidation, the toxins are burnt up and the blood pressure raised. Recovery takes place."

Then they go on, "We are conscious of the danger that exists in first promulgating a hypothesis and then submitting it to experimental investigation, but we venture to seek publication for this paper in the belief that it opens up a new avenue by which the investigation of the disease may be approached and in the hope that it may at least lead to a new line of thought and discussion."

Well, as we all know, these hopes and beliefs were soon fully to be justified. Only three months later, Little and Wright[13] published their preliminary observations on milk fever. They, too, had noticed that the tetany of milk fever presented very similar clinical symptoms to those associated with the lowered calcium content of the blood of parathyroidectomised animals. As the loss of calcium during the later period of pregnancy was known to be considerable, they suggested that at parturition conditions might exist in which the blood calcium content could more easily be lowered.

They therefore estimated the blood calcium of 12 cows affected with milk fever, and after recovery, and were able to show "that considerable diminutions (in mild cases up to 20 to 30 per cent., in severe cases up to 60 per cent.) accompany the onset of this disease." And they pointed out [14] that, though these findings supported the parathyroid deficiency theory of Dryerre and Greig up to a point, the explanation might be more simple—due to the first flow of milk bringing on the loss of calcium from the blood and thus the onset of milk fever.

Though the credit for initial publication of experimental work on what might be called the modern aspect of the milk fever problem must go, therefore, elsewhere, this college may take pride in having had on its staff men of the calibre of Dryerre and Greig who had given a great deal of thought to the problem and who within three years were to publish ample confirmation[7] of Little and Wright's observations and to show that the curative effect of mammary inflation was associated with a rapid rise in blood calcium; and who a few years later, following the work of Little and Wright[14] and Sjollema[19] with calcium chloride injections, were able, in spite of antagonism and criticism, to establish calcium gluconate (later to become borogluconate) therapy as one of the main weapons of the practitioner's armoury[12].

After every great advance there is of necessity a lull, so we are not surprised to find that after the tumult and the shouting of the calcium controversy passed away, a period of quiescence settled down on the milk fever field. Since then the main contributions in this country to the problem have come from Weybridge, where the Biochemical Laboratory, under Dr. H. H. Green, has been carrying out over the last 15 years analyses of blood samples submitted by practitioners. Discussing some of these results, Barker has expressed the opinion[1] that milk fever is not just a simple hypocalcaemia and suggests that any bovine animal showing symptoms of metabolic disorder just prior to parturition or in the following four days has invariably a hypocalcaemia which becomes progressively lower till, somewhere between 5 and 6 mg. per cent., paresis supervenes to be followed by narcosis, coma, or convulsions, depending on whether the blood magnesium is above, within, or below the normal range of 1·8 to 3·2 mg. per 100 ml. respectively. You will perhaps bear with me if I quote rather more fully from Barker's classification as I wish to comment on it later in relation to our own findings. As you may remember, he divides his cases into three main groups:

"(*a*) Where hypocalcaemia is linked with a hypermagnesaemia the patient reels, becomes paretic and narcosis supervenes.

(*b*) Where a hypocalcaemia is linked with a normal magnesium level the patient 'paddles' with the hind legs, becomes recumbent, may or may not get up, finally becomes paretic and coma supervenes.

* Presented to the Scottish Metropolitan Division, N.V.M.A., at Edinburgh, October 20th, 1948.

(*c*) Where the hypocalcaemia is linked with a hypomagnesaemia the patient shows tetany of the fore as well as the hind limbs, hyperaesthesia or may become recumbent, followed by a convulsion."

To allocate cases accurately into those groups demands a fairly comprehensive and reliable history which is not always available, but I have tried to do so in so far as the information at my disposal will permit.

In spite of all the work that has gone on in the last quarter of a century, we would still appear to be no nearer an understanding of the etiology of this disease than in the days of Walley and Schmidt. The hypoparathyroidism theory of Dryerre and Greig, now divested of its " guanidin " component[13], still stands as a reminder of the early speculations of its authors, though it still lacks definite experimental proof.

Numerous other theories have, of course, been suggested. Allcroft[1] holds the view that milk fever is a pathological exaggeration of physiological changes which take place normally at calving, a view to which one could readily subscribe though it does not take one very far. Van der Meulen[15] suggests that the important feature is an inability to replenish the blood calcium from the intestinal tract while Zwynenberg[20] thinks that the basic trouble is the deflection of blood to supply the secretory requirements of the udder, leading to stasis in the peripheral vessels, fall in blood pressure and subsequent endocrine imbalance. More recently, Seekles[18] has suggested that the whole trouble is associated with over-development of the pituitary gland as a result of the forced development of the cow as a milk producer. This is said to cause a complete dysfunction of hormonal regulation.

I am indebted to Dr. Le Roux for a personal communication in which he has elaborated for me the views of this Dutch school of workers, and it would seem that they include auto-intoxication as a connecting factor between hormonal upset and hypocalcaemia.

In spite of all this, we are still not much further forward. In fairness to all, I ought to add that by the end of this paper you will still be no wiser as to the cause of the disease.

Observations

Our observations have been mainly concerned with animals which did not respond favourably to calcium therapy and started in the herd of a friend of mine who had lost four valuable cows which, as indicated in our Clinical Communication[17], had gone down soon after calving with what was diagnosed as milk fever and had failed to get up following repeated subcutaneous injections of calcium borogluconate in relatively large doses (3 to 4 ozs.). The animals became bright and, apparently, normal in every way save for the inability to rise, and though his veterinary surgeon had pinned his faith on time as a sovereign remedy, every one had eventually to be slaughtered; and at the time when he approached me a fifth had just gone down with the same symptoms as before.

Knowing that practitioners occasionally had success with inflation after calcium had failed, I suggested such a procedure, but this was not agreed to—on the grounds that calcium would do all that inflation would do and do it better. The result was that the next animal went the same way as the previous four and simply lay refusing to rise although analysis showed that the blood calcium was rapidly brought back to normal by the treatment adopted.

I then arranged with the farmer and his veterinary surgeon to have periodical blood samples from a number of cows due to calve in the following few weeks and, as indicated already, the main consistent changes noticed were in the calcium and inorganic phosphorus. Table I shows the type of result obtained with animals which calved normally.

These observations were interesting but not original, as Duckworth and Godden[8] had shown in the course of a study of the various calcium fractions in the blood of heifers at parturition that both calcium and phosphorus tended to fall, the calcium very slightly immediately following parturition and the inorganic phosphates more markedly in the 24 hours previous to parturition. In particular, they found that the inorganic phosphorus, which a few days prior to calving was usually about 5 to 6 mgm. per 100 ml., fell without exception at calving time to somewhere in the region

Table I (Cow 14): Blood Composition in Mg. per 100 ml.

Sample number	1	2	3	4	5		6	7	8	9
Date of sampling	24.2.48	26.2.48	1.3.48	4.3.48	7.3.48	Calved 16.3.48, at 5 p.m.	16.3.48	16.3.48	17.3.48	18.3.48
Time of sampling	10.30 p.m.	10 p.m.	10 p.m.	10 p.m.	10 p.m.		7.30 p.m.	10.30 p.m.	8.45 a.m.	8.30 p.m.
Serum calcium	9·8	12·0	10·8	11·0	11·2		—	9·4	8·6	8·8
Serum magnesium	2·523	3·106	3·163	2·549	2·462		2·82	3·079	3·079	3·55
Blood glucose	—	—	—	—	71·0		70·0	70·0	68·0	62·0
Serum inorganic phosphates	5·282	6·58	5·725	5·376	5·508		4·545	4·96	4·14	4·47
Serum sodium	—	352·9	339·7	342·2	358·8		379·1	370·3	360·7	351·1
Serum potassium	—	20·85	—	—	21·34		19·74	18·51	18·51	18·51

Table II (Cow 24—Six-Year-Old, 1,200-Gallon Cow): Blood Composition in Mg. per 100 ml.

Sample number	1	2	3		4	5		6		7	8		9	10	11	12	13
Date of sampling	24.2.48	26.2.48	1.3.48	Calved 24.48, at 8 p.m.	2.3.48	2.3.48	Onset of milk fever	3.3.48	5 oz. borogluconate s/c.	3.3.48	3.3.48	Udder inflated 12.10–12.30; cow rose 12.35	3.3.48	3.3.48	3.3.48	4.3.48	5.3.48
Time of sampling	10.30 p.m.	10 p.m.	10 p.m.		8.15 p.m.	11 p.m.		6.15 a.m.		8.30 a.m.	12 noon		12.35 p.m.	1.30 p.m.	9.30 p.m.	10 p.m.	10 p.m.
Serum calcium	10·4	11·1	9·8		6·6	7·2		5·8		7·4	8·6		10·0	10·6	10·2	8·0	10·0
Serum magnesium	2·922	2·910	3·284		3·717	3·727		3·688		3·163	3·268		3·253	3·163	2·715	2·715	2·695
Blood glucose	—	76·0	118·0		100·0	140·0		—		106·9	30·0		70·0	76·0	80·0	70·0	69·0
Serum inorganic phosphates	6·8	6·726	6·609		No trace of phosphates						4·36		7·076	7·103	8·722	8·524	8·24
Serum sodium	—	344·5	363·3		363·3	377·4		404·7		339·5	344·5		342·7	339·5	348·6	342·4	346·5
Serum chlorides	417·6	408·3	388·3		—	395·6		—		395·6	395·6		—	—	—	—	—
Blood haemoglobin	—	—	10·7		12·3	12·3		—		12·3	10·8		—	—	—	—	—

Table III (Cow 31—Six-Year-Old, 900-Gallon Cow): Blood Composition in Mg. per ml.

Sample number	1	2	3	4		5	6	7	Onset of milk fever	8		9	10	11	12	13
Date of sampling	24.2.48	1.3.48	4.3.48	10.3.48	Calved 11.3.48, at 5 p.m.	11.3.48	11.3.48	12.3.48		12.3.48	Udder inflated	12.3.48	12.3.48	12.3.48	13.3.48	14.3.48
Time of sampling	10.30 p.m.	10 p.m.	8.30 p.m.	10.30 p.m.		7 p.m.	11 p.m.	9 a.m.		1 p.m.		1.30 p.m.	6.30 p.m.	10.30 p.m.	10.30 p.m.	10.30 p.m.
Serum calcium	11·2	11·0	11·2	11·2		7·0	7·0	7·0		5·6		6·8	9·2	9·8	8·8	11·0
Serum magnesium	2·874	2·770	2·155	2·204		2·985	3·079	2·838		2·758		2·748	3·011	2·885	2·419	1·99
Blood glucose	—	—	60·0	56·0		98·0	95·9	56·9		—		—	60·0	59·0	55·0	54·0
Serum inorganic phosphates	6·25	6·522	6·698	6·148		6·15	No trace of phosphates					3·5	6·87	6·69	5·62	5·62
Serum sodium	336·6	340·7	344·0	352·6		356·2	355·2	351·4		350·0		349·7	350·0	345·7	345·0	356·2
Serum potassium	20·73	20·97	20·73	21·73		32·32	33·32	29·61		29·30		35·54	31·85	30·11	23·32	21·96

of 2·5 to 4 mgm. per 100 ml., from which it returned more or less rapidly to normal.

As indicated previously, we were lucky enough to include in our observations two cows, Nos. 24 and 31, which developed milk fever and from these more interesting data were obtained. The history of these animals has already been published[17], but Tables II and III give more details of our findings than was felt desirable to include in a Clinical Communication.

The other spectacular feature was the effect of udder inflation on the inorganic phosphorus which rose at least twice as much as the calcium. As colostrum usually contains much less phosphates than calcium, this observation is rather against the view that inflation acts simply by stopping milk secretion. The magnesium, you will note, did not vary very much over the whole period.

The intriguing feature in each case was the extremely low level of blood inorganic phosphorus during the disease. While this need not necessarily have had anything to do with the lack of response to calcium borogluconate in Cow 24, its rapid rise, following inflation and coincident with complete recovery, was at least suggestive. It is well known that phosphates play an important part in the chemistry of muscle contraction so we began to wonder if hypophosphataemia had been the complicating factor involved in the previous aberrant results of calcium therapy. As we have already indicated, the very next cases of milk fever encountered, however, seemed to negative this idea and so we decided to collect as many blood samples as possible from cows with milk fever before and after treatment and after recovery, in the hope of throwing further light on this problem of the recumbent cow. Accordingly, we arranged with members of the Clinical Department and with one or two outside practitioners to let us have samples and these have slowly been accumulating so that I can now give you the figures from a fair number of random cases, the selection having been mainly on the basis of whether or not it was convenient for the veterinary surgeon to take the samples.

Treatment being entirely in the hands of the practitioner, it has naturally consisted usually of calcium borogluconate injected intravenously or subcutaneously, and in the majority of cases it has been successful.

The next tables give our findings in such cases.

Table IV summarises the history of the cases and you will see that we have had a fairly representative selection, occurring from several weeks before to several weeks after parturition.

TABLE IV

Case No.	Interval between calving and onset	Main clinical signs	Blood analysis (mg. per 100 ml.) Ca	Mg	In. P	Dose of borogluconate	Time between treatment and recovery
4	18 days	Excitement	5·8	0·91	Trace	3½ oz. s.c. + 2oz. $MgSO_4$	5 hours
8	12 hours	Narcosis	6·0	3·63	1·71	3 oz. i.v.	1 hour
9	12 „	„	5·0	2·84	5·52	2 oz. i.v. + 2 oz. s.c.	Few minutes
11	2) „	„	5·6	2·23	1·56	3½ oz. s.c.	Within 6 hours
12	9 „	Coma	3·2	2·78	Trace	3½ oz. i.v.	7 minutes
13	Before calving	Excitement	5·6	2·93	2·05	2 oz. i.v. + 2 oz. s.c.	15 „
14	12 hours	Narcosis	5·0	2·62	2·41	3½ oz. s.c.	Within 12 hours
15	Just before calving	Coma	5·4	2·76	2·21	3½ oz. i.v.	„ ½ hour
17	10 hours	Narcosis	6·6	2·28	2·40	3 oz. i.v. + 1 oz. s.c.	20 minutes
18	Before calving	Alert	6·4	2·29	5·07	—	—
19	Before calving	Coma	5·2	2·44	2·06	4 oz. i.v.	Within ½ hour
21	5 hours	Alert	5·6	2·10	2·34	3½ oz. i.v.	15 minutes
26	21 days	Narcosis	4·2	2·36	1·75	4 oz. i.v.	Within 2 hours
27	7 hours	Excitement	6·2	2·29	1·5	4 oz. i.v.	5 minutes
28 (a)	—	—	6·0	2·58	2·82	2 oz. i.v. + 1 oz. s.c.	Several hours
28 (b)	1½ hours	—	5·0	2·55	2·09	2 oz. i.v. + 1 oz. s.c.	2) minutes
(c)	Just before calving	—	7·8	2·38	2·61	2 oz. i.v. + 1 oz. s.c.	1 hour
32	½ hour	Narcosis	4·95	3·75	2·58	4 oz. i.v.	30 minutes
34	22 hours	„	9·2	1·96	6·22	4 oz. i.v.	—
35	Before calving	Excitement	7·8	2·38	2·61	4 oz. i.v.	—

I have had considerable difficulty in classifying the animals clinically under Barker's headings[3], because several of the animals were quite bright and alert, the only untoward sign being inability to rise. However, I have interpreted "dullness," "sleepiness," and "partial coma" in my colleagues' descriptions as equivalent to narcosis. Those cases which could not be classified in any of Barker's categories I have listed as "alert."

The blood findings, I am afraid, show no great correlation with the clinical picture, but they do bear out the fact that in the great majority of cases of milk fever the inorganic phosphate is low and in about 60 per cent. of our cases it is below the lowest level recorded by Duckworth and Godden in normal healthy cows at parturition.

Treatment was mainly on orthodox lines and the results much as one would have forecast, though a few were rather slow in responding.

The effects of treatment on the blood picture are shown in Table V, which gives the results of analysis of samples taken some time after treatment and where possible, after recovery. You will note in most cases a slight increase in inorganic phosphorus, immediately following treatment, while those cases from which blood samples were obtained after recovery showed on an average a significant rise in this constituent.

TABLE V: BLOOD ANALYSIS (MG. PER 100 ML.).

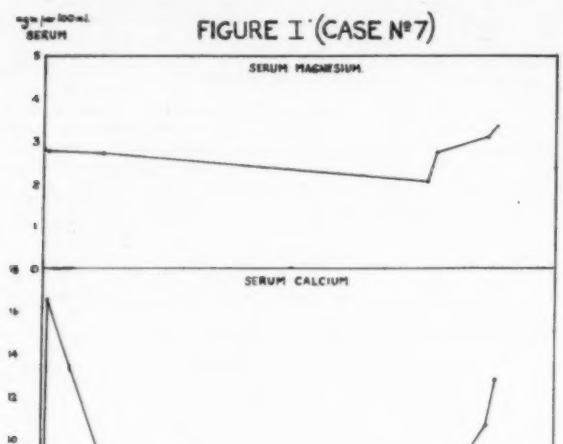
Case No.	Before treatment Ca	Mg	In. P	Soon after treatment Ca	Mg	In. P	After recovery Ca	Mg	In. P
4	5·8	0·91	Trace	6·4	1·69	Trace	10·0	3·43	5·38
8	6·0	3·63	1·71	15·2	3·91	2·56	8·8	3·94	5·61
9	5·0	2·84	5·52	9·2	2·79	5·93	9·2	2·93	5·93
11	5·6	2·23	1·56	6·2	2·29	Trace	—	—	—
12	3·2	2·78	Trace	12·4	2·78	3·6	12·4	2·78	3·6
13	5·6	2·93	2·05	8·0	2·75	2·5	8·0	2·75	2·5
14	5·0	2·62	2·41	5·6	2·57	2·37	—	—	—
15	5·4	2·76	2·21	12·4	2·71	2·39	12·4	2·71	2·39
17	6·6	2·28	2·49	14·4	2·28	2·77	14·4	2·28	2·77
18	6·4	2·29	5·97	—	—	—	—	—	—
19	5·2	2·44	2·06	9·6	2·43	2·13	—	—	—
21	5·6	2·10	2·34	10·6	2·16	2·41	10·6	2·16	2·41
26	4·2	2·36	1·75	13·6	2·35	1·75	—	—	—
27	6·2	2·29	1·5	17·6	2·29	1·6	17·6	2·29	1·6
28 (a)	6·0	2·58	2·82	8·2	2·48	1·34	—	—	—
(b)	5·0	2·55	2·09	11·6	2·55	3·52	11·6	2·55	3·52
(c)	7·8	2·38	2·61	11·6	2·37	2·84	—	—	—
32	4·95	3·75	2·58	13·8	3·35	3·28	13·8	3·35	3·28
34	9·2	1·96	6·22	12·8	1·96	6·73	—	—	—
35	6·8	1·95	1·55	36·0	1·87	1·81	—	—	—
Mean:	5·78	2·48	2·47	11·07	2·50	2·69	11·71	2·83	3·54

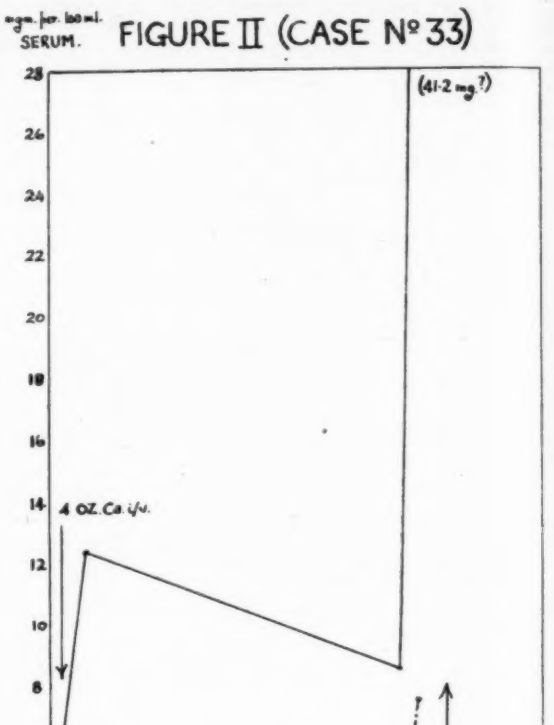
More interesting, possibly, from our immediate point of view are the cases where the response to calcium therapy was not quite so favourable. Most of those have already been described but the following may be of interest.

Case 7 went down about 20 hours after calving and could not rise but lay alert with her head somewhat to one side. Intravenous calcium had her on her feet in about 15 minutes, apparently normal, and the cow was still normal about three hours when the next sample was taken. (Fig. 1 *overleaf*.)

We were re-summoned next day with the news that the cow had gone down again. This time the animal was found to be sweating profusely and extremely excited, and when she endeavoured to rise she fell over backwards. Given 2 oz. calcium gluconate intravenously, she calmed a little, when the udder was inflated. The net result, as you will see, was a marked rise in both calcium and inorganic phosphate. She was left lying quietly and was still the same when we arrived three hours later armed with both calcium and magnesium this time, but got up on stimulation. We gave her the injections, however, and no further relapse was recorded. We found out later that she had had a relapse in between our first and second visits and was given emergency treatment with subcutaneous calcium borogluconate by another practitioner, who was on the premises doing an official tuberculin test, but no figures are available for this phase. The noteworthy feature of this case was the very different clinical syndromes encountered in our two visits, although subsequent analysis showed that the blood calcium, magnesium and inorganic phosphate was practically identical in the two instances.

Case 33 (Fig. 2 *overleaf*) was one of the real old-fashioned type of milk fever with the cow flat out, apparently dead. The cattleman had partially inflated the udder before summoning assistance, but without effect, and when professional assistance arrived, 4 oz. of calcium intravenously failed to get her on her feet though the animal was now conscious. Six hours later she was still dull and the byreman thought she was going to have a relapse. At this point she was given more calcium plus acid sodium phosphate, and though little obvious improvement had occurred when the final sample was taken, she was back to normal within an hour.





Discussion

You will be wondering, doubtless, what conclusions can be drawn from such a diffuse and conflicting mass of data.

Well, in the first place, I think we can agree with those who hold that blood calcium is not by any means the whole story, for there are a number of cases where bringing the blood calcium back to the normal range or even above it failed to effect a cure.

On the other hand, the results do not bear out altogether the thesis that blood magnesium is the deciding factor. One defect of the "hypocalcaemia" explanation of the milk fever syndrome was that it never explained the marked coma of certain cases, and Barker's views on this matter had at least the benefit of simplicity, though with the anaesthetic action of Mg ions in mind, I could never quite understand why narcosis should be associated with a higher level of blood magnesium than complete coma. Be that as it may, I have endeavoured to group our cases on clinical grounds into the categories he gives[2] though, as indicated earlier, it has been necessary to add a fourth group which I have headed "alert" and into which come the majority of our cases which failed to respond normally to calcium therapy. The results are shown in Table VI and they fit in with Barker's theory only to the extent that our two lowest blood magnesiums are in Group 1 and our highest in Group 3 whilst most of Group 2 have a blood magnesium within the normal range. But there are figures within the normal range in all the groups and on an average the figures simply will not fit in with the hypothesis. Statistical analysis of the results confirms that there is no significant difference between the groups. So that if our magnesium figures are to be believed, and I am confident that they are, then in the majority of cases we cannot explain the different symptoms purely on variations in blood magnesium; and incidentally, they do not correlate with the blood calcium or inorganic phosphorus either. One is still in the dark, therefore, as to the reason for this difference in symptomatology.

TABLE VI

1 Excitement		2 Coma		3 Narcosis		4 Alert	
Case No.	Blood Mg.	Case No.	Blood Mg.	Case No.	Blood Mg.	Case No.	Blood Mg.
4	0·91	12	2·78	8	3·63	Cow 24	3·69
13	2·93	15	2·76	9	2·84	Cow 31	2·76
27	2·29	19	2·44	10	2·11	Shamrock	2·79
35	1·95	33	3·47	11	2·23	Pixie	3·27
7 (2nd visit)	2·23			14	2·62	7 (1st visit)	2·82
16	0·81			17	2·28	18	2·2)
				25	2·15	20	2·3
				26	2·36	21	2·1
				32	3·75	23	2·45
				34	1·96	24	2·5
						29	1·31
Mean	1·85		2·86		2·59		2·56

The other constituent in which we have noticed considerable variation is the inorganic phosphorus. Now, of course, we are not the first people to notice a fall in this fraction in milk fever. It was mentioned by Sjollema over 20 years ago[19] and confirmed by Fish[9], who also noted the post-treatment rise with udder inflation, though he records a fall in the first 10 to 20 minutes which we have seldom

observed, and suggests that the return to normal may take a few hours. These observations, however, have tended to be ignored till recent years, possibly partly because they conflicted with the theory of parathyroid deficiency—a phenomenon which is known experimentally to cause a rise in blood phosphate above the normal level. Barker[4] states that there is a lowering of blood phosphate in cases showing tetany, and more recently Allcroft[2] has also stressed the association of low inorganic phosphorus with milk fever, and explains it as a pathological exaggeration of a normal change at calving which is rectified very quickly in response to calcium therapy. We can concur in that view though there would seem to be occasions on which the rectification is somewhat delayed!

Though these phosphate variations have usually been dismissed in the past as being of no great significance, I am not altogether satisfied that such a procedure is completely justified. You may have noticed in the *Record*[3] a few months ago a note on the use of acid sodium phosphate in the treatment of the recumbent cow. This treatment, which recalls the suggestion of Fish[10] that calcium glycerophosphate would be the most logical therapeutic agent to use, was apparently[5] based on observations of Barker, who, I am led to understand, claims that by this means he has practically abolished the problem so far as milk fever in his practice is concerned. While our results do not altogether support the implications of that claim, and in fact some of them are quite against it, others are at least suggestive, particularly in those cases which responded to inflation after calcium had failed to effect complete cure. I am aware, of course, that even in those cases which appear to support the thesis, the rise in phosphate may be coincidental, but nevertheless I cannot help feeling that the whole problem warrants further examination.

This some of my younger colleagues have undertaken to do, and I hope they will have the co-operation and support of members of this Division in their task and that in no far distant time they may be able to come here and present you with a more consistent and more complete story than I have been able to give to-day.

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Discussion

The President (Mr. D. C. Penman), in throwing the meeting open to discussion, remarked that the position seemed to be that while still injecting calcium they should inflate the udder if calcium therapy did not prove effective.

Professor G. F. Boddie: In all sincerity, I welcome this opportunity of congratulating my colleague, Professor Robertson, on the paper he has given us this afternoon; as an opener of the discussion, I had the privilege of a preview of the paper. It is a most interesting exposition of the views he has formed, and I venture to suggest that the biochemical details he has furnished provide a very valuable contribution to our knowledge of the aetiology of those diseases falling into the group designated milk fever. As those who know Professor Robertson would expect, he has been very careful in framing the deductions he has drawn from the biochemical data he has produced.

I need not dwell on the earlier theories of milk fever to which he made brief reference, but I would like to draw your attention again to the statement of Little and Wright in their paper published in June, 1925, that the calcium content of the blood plasma of cows suffering from milk fever showed a diminution of 20 to 30 per cent. in mild cases and of up to 60 per cent. in severe cases, these findings being confirmed by Dryerre and Greig in 1928. According to these criteria most, if not all, of the cases of milk fever described by Professor Robertson were mild in character but yet they failed to make a satisfactory recovery when treated by the parenteral administration of calcium boro-gluconate.

Before we were able to classify milk fever according to its biochemical characters, many of us had realised that, clinically, milk fever was divisible into at least three main groups. First we had what I might term the classical form of the disease—being the form described by teachers and writers; second, we had the form in which there was intense muscular activity, excitement and noise. Third, there were the cases that made some progress towards recovery but remained recumbent for a period dependent on the success attendant on our best endeavour in the realm of symptomatic treatment. Mr. Barker has endeavoured to classify these more scientifically according to the level of the blood magnesium. I have no doubt that for his area Mr. Barker's observations are precise and accurate, but I venture to suggest that milk fever is a variable quantity according to the district, the class of cow and, possibly, the individual farm and its methods of husbandry. In other words, though applicable to the cases in his own area, Mr. Barker's classification may not apply to other areas.

In Table VI Professor Robertson has tried to make his series of cases conform to Mr. Barker's classification. Professor Robertson points out that he cannot explain the different symptoms purely on variations in the blood magnesium. With that statement I entirely agree. In his group showing narcosis there is a blood magnesium of 1·96 mgs., while in his group showing excitement there is a blood magnesium of 2·93 mgs. The mean of the narcosis group is 2·59 mgs.—*i.e.*, those which should have a high magnesium, while the mean of the group showing coma—*i.e.*, those which should have a normal magnesium—is 2·86 mgs. There are no very divergent figures in Table VI that would have disturbed the mean to the extent of producing this contradictory result. I think we are justified in concluding that in Professor Robertson's series of cases the cause of the resistance to treatment is not a disturbance of the blood magnesium.

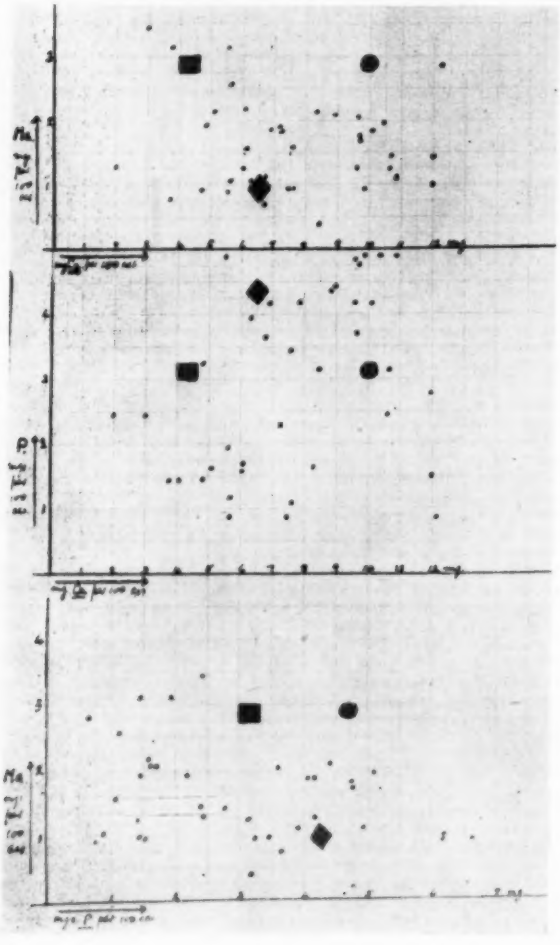
Professor Robertson has brought forward his own evidence that the explanation of this resistance is to be found in the abnormally low inorganic phosphorus, a contention (as he told us) already put forward by other workers.

Before the treatment of milk fever had advanced into the realms of what we might term chemical substitution, all cases, whatever their kind, were successfully treated by udder inflation. Furthermore, lactation, tetany, grass tetany—or as it is now sometimes called hypomagnesaemia—was also treated by udder inflation, and I think the recovery rate by this method of treatment was as high as with the injection of calcium salts. It would seem, therefore, that udder inflation in some way stimulates calcium metabolism, magmesium metabolism and the metabolism of inorganic phosphorus. How is it that udder inflation caused such a rapid recovery in Professor Robertson's cases when the injection of calcium had failed? Indeed, how does udder inflation exert its action in the straightforward case of milk fever? I think the idea that udder inflation stops the loss of calcium into the milk and so leads to an increase in the blood content is quite untenable. In Schmidt's earlier work, using a solution of potassium iodide, the quantity injected was small and it is difficult to visualise that it generated sufficient pressure to inhibit milk secretion. Is it not more reasonable to assume that udder inflation produces an unknown reflex action by virtue of stimulation of the sensory nerve endings in the udder? You will be familiar with Petersen's work on the let-down of milk and how it can be stimulated by the application of warm cloths to the udder. The udder is one of those exquisitely sensitive tissues that when stimulated can produce remote effects possibly reflexly or possibly by the release of hormones. I suggest that if we knew more of what happens when the udder is stimulated we might know more of the fundamental cause of milk fever and of grass tetany. The work of Dryerre and Greig and that of Little and Wright provided a stimulus to biochemists and achieved a great advance in our knowledge of the aetiology of milk fever. Unfortunately, the investigation stopped short of the complete elucidation of the aetiology; that may have been due in part to the success of the calcium treatment of milk fever.

In conclusion, I would like to emphasise the conception that milk fever of cows is, in fact, a group of diseases, each form presenting a somewhat different clinical picture and characterised by biochemical changes peculiar to itself, and that these divergences may be related to the breed, locality and husbandry of the animals concerned. For this conception there does seem now to be available some biochemical basis.

Dr. Stewart welcomed Professor Robertson's very interesting paper since it opened a discussion on those cases of milk fever which did not respond to calcium therapy. At Moredun Institute, with the co-operation of veterinary practitioners, they had examined blood samples from over 70 such cases, and for the purposes of the present discussion he had selected 40 cases in which they had

analysed the blood for calcium, magnesium and phosphorus as well as several other constituents. The results were shown in a Scatter Diagram (Fig. 1) hastily drawn for the meeting.



● Normal
■ M.F.
◆ L.T.

FIG. I.

He thought that there was often confusion in diagnosis and, from the diagram, it would appear that many of the cases diagnosed as milk fever were really cases of lactation tetany, many, indeed, having a low magnesium with a normal calcium, though the majority of this group had a blood calcium in keeping with lactation tetany, viz., 6·6 mgs. approx. Several of these cases had been followed up by bleeding a dozen members of the respective herds, and it was shown that all the herds were in a hypomagnesaemic phase: Dr. Stewart thought that if parturition occurred in such a phase the excitement could reduce the already lowered blood magnesium to a tetany level, as was known to happen occasionally if oestrus occurred in such a phase. He did not think that in typical milk fever the blood magnesium level was altered.

Dr. Stewart showed from the diagram that several of the cases not responding to calcium treatment were similar to those described by Professor Robertson, in that they showed very low levels of inorganic phosphorus in the blood. Several showed a low phosphorus *per se*, but others a low phosphorus combined with a low magnesium or low calcium.

Professor Robertson's remarks regarding the difficulty in assaying such low blood phosphates reminded him of the difficulties they had experienced at Moredun in the measurement of the phosphate content of blood samples obtained from cases of parturient red-water, in which disease the blood phosphate fell to extremely low levels. He wondered if there could be any connection between the aetiology of those cases of milk fever with the low blood phosphorus and that of parturient red-water, which occurred at 10 to 20 days after parturition with a very similar blood phosphate status, or if blowing up the udder would raise the blood phosphorus in that condition, especially since they were having trouble in finding a phosphorus salt completely suitable for intravenous injection.

He had only one remark to make on Professor Boddie's contribution and that was concerning the suggestion that udder inflation would cause a rise in blood magnesium. He could not recall any paper in which this was stated and it was not in accordance with his own experience, inasmuch as inflation of the udder did not bring recumbent cases of lactation tetany to their feet.

He thought that Professor Robertson's paper had been well worth while as it had reopened the whole question of the aetiology of milk fever.

Mr. J. Milne: He would like to ask Professor Robertson if fatty liver infiltration was ever a complication of milk fever. He had encountered several cases which commenced as ordinary milk fever—these were often fleshy cows. However, they failed to respond to calcium and inflation; they would sit up for a while after calcium was injected, but later they became stupid and lay stretched out. Some of these cows, when slaughtered, showed fatty types of liver. Some of them had been given glucose with some success.

Dr. Dryerre: He thought the paper admirable and very interesting. He was interested in the aetiology of milk fever, and recalled his early differences of opinion with Fish, who stressed he importance of blood phosphorus. Professor Boddie, he thought, had probably explained these differences of opinion when he said the signs and symptoms might vary with the district and husbandry. Professor Robertson's experiences of failure of calcium therapy and success with inflation emphasised an important point. Lactose, a product of mammary gland activity, appeared in the blood after inflation. Consequently, one might argue that perhaps inflation put something else from the udder into the blood which might affect recovery. With regard to Mr. Milne's remarks referring to fatty livers in cases of milk fever not recovering, Dr. Dryerre wondered if Mr. Milne had investigated the livers of cows dead soon after parturition, but which were not affected with milk fever. When carrying out researches a number of years ago along with Professor Robertson, he had found that fatty livers were common in pregnant ewes, and there was a progressive fatty infiltration of the liver during pregnancy. He considered the fatty livers referred to by Mr. Milne did not mean much unless normal cows at the end of pregnancy had been examined and found to be free from fatty infiltration. Dr. Dryerre had also noticed the low blood magnesium levels in cows turned out to pasture; he did not know the cause —as the pastures were rich in magnesium. In an experiment where he fed small quantities of magnesium salts he had found the animal excreted more magnesium than it took in, and this was associated with a fall in blood magnesium. He wondered what caused this negative magnesium balance.

Dr. Russell Greig: Professor Robertson's address had brought back to him many memories of the exciting and happy days long ago when Professor Dryerre and he were closely engaged on their milk fever research. He had been somewhat put to the blush to hear retailed some of their early views on the aetiology of milk fever. Dryerre and he had abandoned the guanidin theory at an early stage. Professor Robertson had mentioned that Schmidt originally used a very weak solution of potassium iodide in his udder injections. The solution used in this country was even much weaker than that recommended by Schmidt. When Schmidt's paper was published in *The Veterinary Record* the translator misinterpreted the gramme symbol "g." as meaning grains, so that the solution used in Britain was only one-fifteenth the concentration recommended by Schmidt. Since iodide of potassium was a very weak antiseptic at best, and since the treatment produced such spectacular cures, it was somewhat surprising that the belief that the injections acted by producing an antiseptic effect in the udder should have persisted so long. Dr. Greig was not clear as to Professor Robertson's criterion of cure. It was certainly true that complete cure could be regarded as achieved when the cow regained her feet and became in every way normal, but the hypocalcaemia might be completely overcome although the cow did not rise. In several instances cows persisted in remaining recumbent for some considerable time after cure had been effected and might rise only when they received some stimulus such as being startled by fright, etc. It would be misleading, therefore, to suppose that the calcium imbalance had not been righted until the animal had herself demonstrated that she could regain her feet. It might be true that milk fever was only one of a group of conditions which could be labelled "the milk fever complex," but he thought it was generally agreed that milk fever was a definite pathological entity, the immediate cause of which was an acute

hypocalcaemia. If the condition was one of a group, then hypocalcaemia was common to all the members of the group. It could be said that the cause of milk fever was unknown in so far that the cause of the hypocalcaemia was not certainly known, but if it were demonstrated that the cause of the hypocalcaemia was, say, a parathyroid dysfunction, we would have to ask what was the cause of this parathyroid dysfunction. If in the future that were demonstrated to be due to, say, a dysfunction of some other group of glands, there would then arise the question what was the cause of the dysfunction of this or that other group of glands, and so on *ad infinitum*. Such considerations applied to metabolic diseases in general. He could visualise discussion on the subject for many years ahead before the primary cause ultimately would be found. Meanwhile, the essential, immediate, underlying cause had been demonstrated and a specific method of rapid and complete cure by calcium therapy had been evolved. He congratulated Professor Robertson for having reopened the subject.

Major Linton Robertson asked Professor Robertson if he had ever met milk fever after a difficult parturition. He himself had never encountered it in such circumstances.

Mr. R. G. Anderson: He had seen good results following udder inflation and believed it still seemed to be the best line of treatment. He regarded the disease as being one of the circulation and not of the nervous system. He had seen cows with milk fever die following the relieving of a distended bladder or intestine, and this also might be a pressure effect.

Mr. R. F. Hall: He noted that Mr. Milne sometimes gave glucose in a difficult case. He had also thought that 2 or 3 ozs. of glucose along with calcium seemed to help in certain cases. He would like to hear Professor Robertson's views on this.

Mr. Begg: He had never seen a cow rise within 10 to 15 minutes after inflation; nor had he ever lifted a cow by using inflation following the failure of calcium, but he had succeeded in doing the reverse. Mr. Begg considered the cow was a hypochondriac—sometimes he had found that if a cow would not rise following a second injection of calcium, if all the other cows were let out of the byre she would then get up; he had also experienced cows getting up following their being pulled into the float which was to take them to the abattoir. He usually gave 3 to 4 ozs. of calcium intravenously, followed by 3 ozs. subcutaneously, and nine out of ten cases rose to their feet within ten minutes. If not successful, he repeated the calcium injections in three hours. The odd cow which seemed to be rather dull he pulled outside, and this might cause the cow to rise. He maintained that inflation never improved such cases. He had come across cows in oestrus, or more often which had been in oestrus 12 hours before, taking fits, showing tetany, sweating, foaming at the mouth and exhibiting muscular spasms, where in every case blood magnesium was low (0·8 and 0·9 mgs. per cent.) and he treated such cases with magnesium. However, he had found that injections of calcium caused the magnesium to rise also.

The licking maniac cow he found had also a low magnesium level in its blood, but calcium would cure such cases in three or four hours.

The Reply

Professor Robertson, in reply, said that Professor Boddie had credited him with rather more certainty about the significance of the hypophosphataemia than he really possessed. What he did suggest was that the problem warranted further study. Professor Boddie's idea that milk fever was really a group of several diseases was probably right; it was certainly a variable quantity, for there were instances of practitioners suddenly meeting with trouble after having used calcium therapy successfully for years. On the other hand, there was no question in such cases of the variation being associated with a difference in district, breed or system of management. As to how inflation worked, he agreed with Professor Boddie that it could not be simply a case of putting milk constituents back into the blood or of stopping secretion. The animals they had inflated had been completely milked out before inflation. Moreover, the fact that blood phosphates rose twice as much as calcium while colostrum usually contained only half as much, was against the explanation being a simple stoppage of secretion. He noted that Professor Boddie suggested inflation might have a reflex stimulating effect and that would certainly be a very interesting line of research.

Dr. Stewart's cases not responding to calcium were interesting. Dr. Stewart said some of these cases were lactation tetany. Professor Robertson believed that one of his cases which did not respond to calcium was also this condition. He was particularly interested in Dr. Stewart's remarks on parturient red-water and was glad to note that others had also been perturbed by an apparent complete absence of blood inorganic phosphorus. Like Dr. Stewart, he had never seen much change in blood magnesium as the result of inflation.

Mr. Milne had really been answered by Dr. Dryerre, who emphasised that there was a marked fatty infiltration of the liver during pregnancy. When one took into account the fact, which he had verified experimentally with pregnant sheep, that fasting would also produce fatty infiltration, particularly towards the end of pregnancy, there was little difficulty in explaining Mr. Milne's findings, but he doubted if they were the real explanation of the lack of response to treatment. The response to glucose, however, was interesting and suggested that probably something of the acetonaemia type was present.

Dr. Dryerre's remark about feeding small quantities of magnesium leading to magnesium imbalance and a deficiency of magnesium in the blood was interesting; on the other hand, they had gone into the diet in many of their milk fever cases but could not find any direct association between feeding and the occurrence of the condition. Dr. Dryerre's suggestion that inflation might be returning some other important constituent as yet unsuspected to the blood gained some support from the fact that they had found that differences in symptomatology could not be explained solely on the basis of either calcium, magnesium or phosphate.

Dr. Greig's information about the mistranslation of Schmidt's paper was new to him—apparently the strength of the potassium iodide solution had not mattered and yet our ancestors certainly got results with this solution. Dr. Greig had asked him a difficult question with regard to the criterion of cure. He regarded an animal cured when it was back on its feet, eating, drinking and milking normally and behaving generally as a cow should. Dr. Greig, apparently, considered a cow cured even if she did not regain her feet, provided the blood calcium was back to normal, but he was afraid the farmer would not think much of this idea. Dr. Greig had also made the point that the further we probed the deeper we would have to go, so that we would never really know the ultimate cause. That might be true in essence, but looked at practically, it was really a counsel of despair. If he knew enough of the underlying causes to be able to prevent the condition, he would be satisfied and leave the quest for ultimate causation to philosophers, divines and similar intellectuals who were doubtless adept at such matters.

Major Linton Robertson had asked if he had ever encountered milk fever after a difficult parturition. In their records, out of a series of 36 cases, one followed mild dystokia possibly caused by uterine inertia, and another occurred after marked dystokia.

Major Anderson's remarks with regard to circulatory upset were interesting and recalled the fact he had already mentioned that Zwynenberg believed a lowered blood pressure to be the fundamental cause. In the days before a successful treatment was available there was a fairly widespread belief that the condition could be prevented by a gradual emptying of the udder during the first few days after parturition; and it certainly was plausible to assume that inflation counteracted the fall in pressure caused by a too sudden emptying of the distended mammary gland.

Professor Robertson said he must just disagree with Mr. Begg with regard to the effects of inflation, as he had seen with his own eyes in the presence of reliable witnesses, cows which had not responded to calcium therapy get up almost immediately following inflation. Mr. Begg's theory of cows being hypochondriacs was not new and indeed, one of his colleagues frequently put lack of response to treatment down to the "psychology" of the cow. Mr. Begg made the point that the dosage of calcium had been inadequate and that the subcutaneous route was not satisfactory. But they must remember that this same treatment had been used with success on the same farm for a long number of years and then suddenly five cows in succession failed to respond to it. He had instances of other practitioners meeting with similar experiences. It had been suggested, too, that they had not used adequate stimulation, but he could assure Mr. Begg that they had tried all that he had suggested—in fact, they had done everything short of lighting a fire under the cow's tail! They had even got the float to take them to the butcher—and four of them went!

With regard to Mr. Hall's success using glucose, as calcium had also been given he did not know how much credit to give glucose.

In conclusion, he would like to say that the work presented to them that day had been the product of the activity of a number of individuals and that he had simply acted as spokesman. Credit, if indeed any was due, must go largely to his various colleagues and assistants who had done the bulk of the work though he had been privileged to act as co-ordinator and mentor.

Weekly Wisdom

I am entirely convinced that what is more than anything else lacking in the life of the average well-intentioned man of to-day is the reflective mood.—Arnold Bennett.

The Transport of Animals

In response to a request from the South Wales Division, the following notes have been prepared, drawing attention to the present legal position with regard to the carriage of animals under the provisions of the Diseases of Animals Acts.

It should be noted that although local authorities are required to execute and enforce many of the provisions of the Diseases of Animals Acts and the numerous Orders made thereunder, and to appoint lay inspectors for this purpose, this does not preclude others taking *legal action* with regard to infringements of these enactments, and the rights of common informers in this respect were established in a High Court case as far back as 1896. (*Regina v. Stewart*—60 J.P. 356.) Thus the R.S.P.C.A., or any member of the public, may institute proceedings, although normally the matter would be reported to the local authority inspector for action. In most areas, the police act as local authority inspectors under the Diseases of Animals Acts, but this is not invariably the case, a notable exception being in the London Metropolitan and City Police Divisions where each local authority appoints inspectors, who are not police officers.

The enactments governing the transport of animals are the Transit of Animals Orders, 1927–39 (which relate to cattle, sheep, goats, all other ruminating animals and swine), the Exportation and Transit of Horses, Asses and Mules Orders of 1921–37 (which apply, in general, the main provisions of the Transit of Animals Orders to horses, etc.), and the Conveyance of Live Poultry Order of 1919 and the Poultry (Exposure for Sale) Order of 1937 (which relate to domestic fowls, turkeys, geese, ducks, guinea-fowls). It should be noted that although the Conveyance of Live Poultry Order applies to pigeons, the Poultry (Exposure for Sale) Order does not.

The Transit of Animals Order of 1927 deals with the conveyance of cattle, sheep, goats and swine by water and rail, and the Order of 1931 by *road transport*.

Water Transport

Water transport applies to vessels carrying animals from any port in Great Britain to any other port in Great Britain, Ireland, the Channel Isles or the Isle of Man. Animals can only be carried on such *vessels properly fitted* for the purpose and detailed provisions are given as to the parts of the vessels that may be used for animals, the pens and fittings of the vessels, passageways, ventilation and light. Provision must be made for *food and water* when the voyage is to take more than 18 hours. All bulls, horned cattle, fat cows and heifers, pregnant cattle and milch cows must be properly *secured by head ropes* and mixed consignments must be divided into various classes and *separately penned*. *Sheep shorn within 60 days* may not be carried on exposed decks between November 1st and April 30th.

These provisions do not apply to ferry boats or boats plying on a river, canal or other inland water, but the fittings shall be such as to protect the animals from injury and unnecessary suffering as far as practicable and the decks must be strewn with sand, etc., to prevent slipping.

Monthly returns have to be submitted to the Ministry concerning animals which have died or been injured or calved during transit and the numbers which have been carried on the vessel.

Overcrowding is prohibited and only *goads* of approved pattern are allowed to be used. No cow is permitted to be embarked on a vessel if the *calving of the cow* on the vessel or on landing is reasonably probable.

No animals shall be permitted to be embarked on a vessel if, owing to *infirmity, illness, fatigue*, or any other cause, it cannot be *carried without unnecessary suffering* during the intended passage.

Provision is made for *food and water* at shipping and unshipping places. Where a *diseased animal* is landed at any place, that place shall not be used for other animals liable to be infected, until it has been as far as is practicable cleansed and disinfected to the satisfaction of an inspector.

Where a *breach of these provisions* occurs, the owner, charterer, master—and the owner lessee and occupier of the place where the animals are put on board or landed—and also in the case of overcrowding of a vessel in any part or pen, the consignor of the animals shall each be guilty of an offence against the Act of 1894.

Carriage by Railway

Every railway *cattle truck* shall be fitted with battens or *proper foot-holds* or shall be strewn with litter or sand, etc., and shall be fitted with a roof and with falling loading doors and all internal projections shall be rounded.

All bulls, whether polled or not, must be securely tied up by head or neck and all horned stock carried in the same railway truck with a bull, unless separated therefrom by a suitable partition, must also be tied by the head or neck.

No cow shall be permitted by the owner thereof, or his agent, or the consignor or any person in charge thereof, to be carried by railway if the *calving of the cow during transit* by railway is reasonably probable.

No animal shall be permitted to be carried by railway if, owing *to infirmity, illness, fatigue*, or any other cause, it cannot be carried without unnecessary suffering during the intended transit by railway.

It is provided that *calves, sheep, goats and swine*, if carried in the same railway truck or other railway vehicle with any head of cattle (other than a calf), or a horse, ass or mule, shall be separated therefrom by a suitable partition, but this provision shall not apply to the conveyance of a *cow with its unweaned calf*, if they are separated from other animals.

A railway company shall not allow any railway truck, or other railway vehicle or any compartment thereof used for carrying animals on the railway to *be overcrowded* so as to cause injury or unnecessary suffering to the animals therein.

Section 23 of the Act of 1894 is amended by extending the maximum period during which these animals may be kept without a *supply of water* from 24 hours to 36 hours in the case of sheep and 27 hours in the case of cattle and swine.

At every railway station at which animals are habitually loaded, unloaded or detained during transit, the railway company or companies concerned shall make a provision of water to the satisfaction of the Minister for the animals carried, or about to be or having been carried, on the railway, and such provision of water shall be easily accessible to the animals so carried.

Where a *breach of these provisions* occurs the owner of the animal or his agent—the consignor—the railway company carrying the animals, or owning or working the railway on which they are carried —shall, each in respect of his or their own acts or omissions, be guilty of an offence.

It is provided that where an inspector is of the opinion that an animal intended to be carried by vessel, rail, or road cannot, owing to *infirmity, illness, injury, fatigue*, or any other cause, be carried or conveyed without unnecessary suffering, or that the *calving of the animal* during transit is reasonably probable, he must serve a notice to that effect on the person in charge of the animal and until such notice is withdrawn it shall be an offence to carry the animal.

Road Transport

The Order of 1931 deals mainly with the cleansing and disinfection of vehicles and the keeping of records of stock carried by road transport. There are also detailed provisions as to the construction of road vehicles.

It is worthy of note that the Order as to the construction of motor vehicles applies only to vehicles used (*a*) for hire for the conveyance of animals, or (*b*) for the conveyance of animals the property of a dealer, or for the conveyance of animals in connection with the trade or business of a dealer, or (*c*) for the conveyance of animals belonging to two or more persons. A farmer, for instance, who conveys his sheep in his own motor vehicle from one farm to another for the purpose of grazing, would not, apparently, come within the provisions.

Motor vehicles used for the conveyance of animals must be provided with battens or some suitable means of foothold—the interior must be free from projections—capable of permitting a protective roof to be provided—admit of inspection of the inside from outside —be adequately ventilated and be provided with ramps, side rails, etc.

The provisions made as to the carrying by road of unfit animals or cows in calf, the securing of cattle in road vehicles, the separation of mixed consignments and provisions as to overcrowding, are similar to those already outlined when carrying animals by vessel or by rail, but the Order does not prohibit the carriage of an unfit animal by road in a vehicle being used as an ambulance.

The Exportation and Transit of Horses, Asses and Mules Orders

Prohibit the exportation of horses, etc., to the Continent of Europe except from certain specified ports and only if certified by a veterinary inspector appointed by the Ministry to be of a certain minimum value, not over eight years of age, and capable of being conveyed by sea and being worked without suffering.

Provisions are laid down for the fittings of vessels and the methods of securing and conveying the animals.

Carriage by Railway and by Road.—Provision on somewhat similar lines to that laid down for the carriage of other animals in the Transit of Animals Orders is made in these Orders with respect to horses,

(Concluded at foot of next column)

CLINICAL COMMUNICATION

Enteritis of a White Rhinoceros Associated with Pseudomonas pyocyanea Infection

J. K. THOMSON and F. W. PRIESTLEY,
Sudan Veterinary Service,
and
J. B. POLDING,
Khartoum Veterinary School.

On January 17th, 1949, a white rhinoceros, approximately two months old, found near Tonj in the southern Sudan, was flown to Khartoum, the intention being to rear it for export.

The animal was housed, in isolation, in a mud-built hut, given a plentiful supply of grass bedding and ultimately covered at night with a blanket. The latter precaution was taken because the night temperature in Khartoum (55° F.) was then considerably lower than that in the south (64° F.) and the animal clearly disliked these relatively cold nights.

The diet, consisting of diluted (2:1) boiled cow's milk with an addition of lactose, was given every two hours from 6 a.m. to 8 p.m. by means of a bottle and teat. Orange juice and radio-malt also were given daily.

During the first eight days after its arrival in Khartoum the rhinoceros appeared in good health. It took its food readily and the passage of faeces was normal and regular. The rectal temperature remained almost constant between 100° and 101° F. On the ninth day it started cutting its molar teeth and then resented the introduction of the teat, but in less than 48 hours it was apparently normal again and fed greedily. However, early on the morning of the 29th it was found unable to stand and it died at 8.30 a.m.

At autopsy it was noted that the internal blood vessels were engorged. In the lungs there were a few haemorrhagic infarcts measuring 3 to 4 inches across, and in the liver there were signs of incipient fatty changes. The chief abnormality was confined to the intestines. The whole of the intestinal tract was inflamed and there were one or two haemorrhagic areas in the duodenum; the mesenteric lymph glands were hyperaemic.

Microscopic examinations of the blood and lung infarcts were negative for any form of micro-organism. Wet films of the intestinal contents showed no protozoon parasites but enormous numbers of very actively motile, Gram-negative bacilli. Bacteriological examination of the intestinal contents showed that these bacilli were present in almost pure culture. The organism fermented glucose without gas production but had no action on lactose. On agar it produced a typical diffusible green pigment. It was therefore classified as *Ps. pyocyanea*.

Since this organism was present in the inflamed intestine in virtually pure culture it seems probable that it was the cause of the enteritis and ultimately of death. It would seem likely that the rapid transport from a relatively warm to a relatively cold climate had so lowered the animal's resistance that an organism, perhaps normally a commensal, of mild pathogenicity had been able to proliferate and cause disease.

Summary

A case of enteritis associated with *Ps. pyocyanea* in a young white rhinoceros is described.

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etc., to ensure the feeding and watering of these animals during transit and to prohibit overcrowding and the carriage of unfit animals.

Conveyance of Live Poultry Order.

Provisions are made for conveyance of poultry by sea, road and rail. Crates, etc., must be so constructed and the number of birds they contain must be such as not to cause unnecessary suffering. Exposure to bad weather and excessive heat is prohibited and the crates and birds must be handled in a humane manner. It is illegal to convey birds if unnecessary suffering is caused through infirmity or disease.

The Poultry (Exposure for Sale) Order

This Order prohibits the exposure for sale if, owing to illness, infirmity, injury, fatigue or any other cause, unnecessary suffering is likely to result, and an inspector may order the removal of poultry for treatment or destruction.

ABSTRACTS

[Technique of Direct Blood Transfusions in Large Animals. Coffey, W. M. (1949.) *Vet. Med.* **44.** 113.]

In large-animal practice transfusion of whole blood is valuable in several conditions including shock, haemorrhage, purpuras, anaemias and icterus in foals. Typing seems advisable only in the thoroughbred animal or in the icteric foal, which condition may be due to some Rhesus-like factor when the dam should never be considered as a donor.

Donor and recipient stand side by side. A continuous flow ("Shikles") syringe with 3 ft. 6 in. of tubing on the inlet and outlet connections is flushed with 10 per cent. sodium citrate solution. The needle on the inlet tube is then inserted into the jugular vein of the donor and the apparatus filled with blood to exclude air before commencing the transfusion.

In the icteric foal where large volumes of blood may have to be given it may be necessary to drain blood from its opposite jugular at the same time.

J. A. L.

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[Cobalt and Copper in the Nutrition of Sheep. Marston, H. R. *et al.* (1948.) *J. Agric. Sci.* **38.** 216-228. (1 plate, 3 figs., 3 tables.).]

Part I. Experiments are described which extend the previous work (Marston *et al*) on coast disease of sheep. It was shown that the nutritive quality of the fodder indigenous to the shell-sand littoral of South Australia is limited by the low cobalt and copper content. Over a period of six years a supplement of Co. and Cu. alone prevented coast disease, the animals withstanding three successive pregnancies and producing normal lambs. Initially 2 mg. Co. + 2 mg. Cu. were administered thrice weekly, but after two years the dose of Cu. was increased to the equivalent of 7 mg. Cu./day. There were no observable results from feeding Fe., Mn., Zn., or Ni., as a supplement, indicating that the pastures were able to supply these minerals in adequate amounts.

The untreated animals and those receiving Cu. supplement only showed cobalt deficiency; they declined steadily and died within a year. The symptoms were aplastic anaemia, serous exudate from eyes, greenish hue and fragility of the skin and lethargy.

Those on Co. supplement alone exhibited copper deficiency. The symptoms and time of onset were variable but most died within the second year.

Part II. This experiment was designed to study the degrees of copper deficiency. The first experiment indicated complete depletion of all copper reserves after two years and that 1 mg. Cu./day was insufficient. Administration of supplements was by mouth, by syringe thrice weekly.

(1) The untreated group and those receiving 10 mg. Cu./day developed typical symptoms of cobalt deficiency and most died within a year; (2) those receiving 1 mg. Co. + 10 mg. Cu./day remained healthy; (3) those receiving 1 mg. Co./day developed hypochromic anaemia after 28 weeks, and retardation of growth became progressively worse over the four years. After 2½ years ewes in this group produced ataxic lambs; (4) those receiving 1 mg. Co. + 1 mg. Cu./day had a similar course, but the symptoms were delayed; (5) those with 1 mg. Co. + 5 mg. Cu./day grew well and showed no anaemia, but after two years they showed abnormal keratinisation of the wool as the first symptom of copper deficiency.

P. K.

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[The Effects of Copper Deficiency and of Chronic Over-dosage with Copper in Border-Leicester and Merino Sheep. Marston, H. R., & Lee, H. J. (1948.) *J. Agric. Sci.* **38.** 229-241. (4 plates, 2 figs., 10 tables.).]

The effects of copper deficiency and excess were studied over a period of 3½ years, on pasture deficient in cobalt and copper. Each group received thrice weekly doses equivalent to 1 mg. Co./day. The copper dosage was varied, *viz.*, nil, 1, 5, 50, 100 mg./day.

(1) Those receiving Co. alone showed copper deficiency, *i.e.*, anaemia, loss of weight, the lambs were generally weak and some exhibited ataxia; (2) 1 mg. Cu./day delayed the onset of symptoms, but was insufficient to prevent ataxia occurring in the lambs of ewes after two years on the supplement; (3) the supplement of 5 mg. Cu./day proved sufficient and lambing was uneventful in this group.

The first symptom of copper deficiency is a marked deterioration in the quality of the fleece. The characteristic crimp becomes progressively less distinct and the super-imposition of secondary waves occurs. The effects were noticed in the untreated and 1 mg.

Cu. groups. After two years it was noticed in the 5 mg. Cu. group, indicating that 5 mg. was an insufficient supplement. (A number of plates show this phenomenon very clearly.)

The two breeds showed different reactions to excessive copper dosage. A supplement equivalent to 100 mg./day produced crises with haemolysis and icterus. The Border-Leicesters quickly succumbed. In the Merinos these crises were not always fatal; one-third of them showed no symptoms as a result of 100 mg. Cu./day. A dosage of 50 mg. Cu./day produced crises in one-third of the sheep, but recovery was the rule. In the remainder no observed crises occurred, and the fleece of these sheep was "stylishly crimped" throughout the 3½ years.

P. K.

REVIEW

[Dictionary of Scientific Terms. By I. F. HENDERSON, M.A., and W. D. HENDERSON, M.A., B.SC., PH.D., F.R.S.E. Fourth Edition by JOHN H. KENNETH, M.A., PH.D., F.R.S.E. (1949.) Pp. 480+xvi. Price 32s. net. Oliver & Boyd: Tweedale Court, Edinburgh; 98, Great Russell Street, London, W.C.]

In bringing out the fourth edition of this excellent dictionary of terms used in biological literature (again admirable in format and typography) in conformity with the schedule of approximately decadal issue which has been maintained since the first edition in 1920, author and publishers merit high praise. The text of the third edition has been revised considerably and some 2,000 terms have been added. Items of systematic nomenclature such as genera, orders, and species necessarily have been omitted. Two great and obvious difficulties have been overcome successfully: the exacting task of maintaining so high a standard of production in these difficult days—and here the willing co-operation of the publishers in resisting the temptation to issue the new matter as a supplement is gratefully to be acknowledged—and that presented by the almost overwhelming increase in scientific terminology inseparable from advances in the fields to which the volume relates: biology, botany, zoology, anatomy, cytology, embryology and physiology. In this connection the selective quality of the work of the compiler is seen to great advantage, enabling the book usefully to fulfil its function of making available to students and other readers of the Biological Sciences innumerable terms which, while in common use amongst the devotees, are not to be found in the usual handy-sized dictionary. Pronunciation, derivation and definition are, of course, covered, though the first admittedly often is debatable, as pronunciation is subject to continual fluctuations. The number of cross-references has been augmented to a considerable extent, and helpful adjuncts are lists of sound symbols used in pronunciation, of abbreviations and of equivalent units of measurement.

Though the veterinary interest of the Dictionary is, as it were, mainly collateral, the volume is one which the professional man, of scientific calling, or bent, will be well advised to have at his elbow.

ANIMAL HEALTH TRUST'S MOBILE CARAVAN

Visitors to Agricultural shows this season will see a new feature —the Animal Health Trust's Mobile Laboratory, in the shape of a caravan equipped for scientific investigation and also as an enquiry bureau for those interested in the Trust's work.

The Trust has equipped this unit as a self-contained laboratory, which can be used to visit areas affected by specific disease problems; and also to travel the country visiting show-grounds and other centres of livestock interest, where a service of information on the Trust's activities will be useful. The caravan provides sleeping accommodation for the scientific staff when they are on a job. It will also, of course, be a mobile advertisement for a cause which the farming community know well to be a sound and developing contribution to better animal health in this country.

The shows at which the Mobile Laboratory will be on exhibition are:—

Show	*Place*	*Date*
Three Counties.	Worcester.	June 14th to 16th.
Royal Counties.	Reading.	June 22nd to 25th.
Norfolk and Norwich.	Norwich.	June 29th to 30th.
Royal.	Shrewsbury.	July 5th to 8th.
Kent.	Maidstone.	July 13th to 14th.
Yorkshire.	Wakefield.	July 26th to 28th.
Rumney Horse Show	Nr. Cardiff.	August 1st.
Royal Welsh.	Swansea.	August 10th to 13th.
Royal Lancs.	Blackpool.	August 16th to 19th.

NOTES AND NEWS

Diary of Events

June 13th.—Emergency Meeting of Lincolnshire and District V.M.A., Institute of Animal Pathology, Milton Road, Cambridge at 2.30 p.m.

June 14th—Scientific Meeting of the Zoological Society of London, at Regent's Park, N.W., 5 p.m.

June 15th.—Annual General Meeting of Lincolnshire and District V.M.A., at Saracens Head Hotel, Lincoln, 1 p.m.

June 15th.—Meetings of the N.V.M.A. Editorial Executive Committee, at 36, Gordon Square, W.C.1, 2 p.m.; Main Committee, 3 p.m. (Note alteration from 2nd to 3rd week in the month).

June 16th.—Summer Meeting of the Lancashire Division, N.V.M.A., at the County Institute of Agriculture, Hutton, nr. Preston.

June 17th.—Meeting of the Biochemical Society at Nottingham (Department of Biochemistry, the University) 2 p.m.

June 22nd to 24th.—June Meetings of N.V.M.A. Council and Committees in Edinburgh:

June 22nd.—Organising Committee, 2 p.m.; Home Appointments Committee, 4 p.m.

June 23rd.—Veterinary State Medicine Committee, 10 a.m.; General Purposes and Finance Committee, 2 p.m.

June 23rd.—Meeting of Western Counties Division, Royal Clarence Hotel, Exeter, 2 p.m.

June 24th.—Parliamentary and Public Relations Committee, 10 a.m.; **Meeting of Council**, 11.30 a.m.

June 21st to 24th.—Royal Highland and Agricultural Society's Show at Dundee.

June 23rd and 24th.—R.C.V.S. Membership Written Examination.

Aug. 8th to 13th.—Fourteenth International Veterinary Congress, Central Hall, Westminster, and Church House, Westminster, London, S.W.1.

Aug. 12th to 15th.—Sixty-seventh Annual General Meeting and Congress, N.V.M.A., in London.

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First Meeting of the Reconstituted Council of the Royal College of Veterinary Surgeons

The first meeting of the reconstituted Council of the Royal College of Veterinary Surgeons was held at the Royal College at 2.30 p.m. on Wednesday, June 1st, 1949.

The report of the Selection Committee having been read, the Council proceeded to the election of a President.

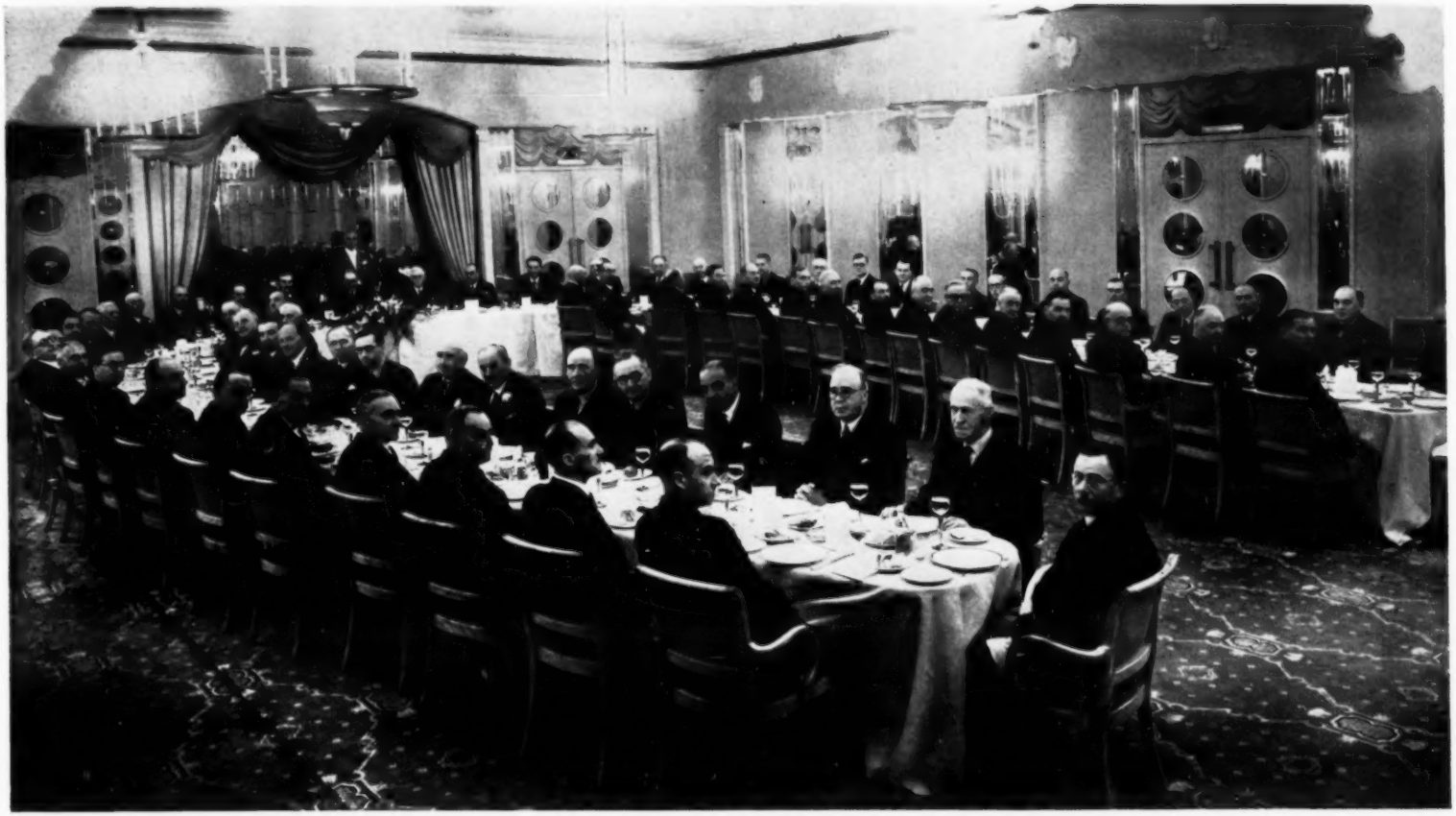
The unanimous recommendation of the Selection Committee was that Professor T. Dalling, M.A., M.R.C.V.S., F.R.S.E., Chief Veterinary Officer, Ministry of Agriculture and Fisheries, be appointed President for the year 1949-50. This recommendation was approved by the Council and in the scene of solemn ceremony the outgoing President, Mr. Howard Dawes, installed Professor Dalling as the new President.

Professor Dalling, in thanking the Council, made a moving speech in which he referred to the many previous occupants of the Presidential chair whose names were inscribed on the board in the Council Chamber and whose portraits decorated the walls of the Royal College. He stated that he felt keenly the privilege of being President on this very historic occasion.

After the installation of the President the Council proceeded to the election of the Vice-Presidents.

It was pointed out that when the new Royal Charter was granted it would be possible for the Council to elect more than two Vice-Presidents. It was therefore unanimously decided to elect Mr. Howard Dawes, the immediate Past-President, as senior Vice-President for the year 1949-50 and to elect as junior Vice-Presidents Professor J. G. Wright, Mr. J. F. D. Tutt, Captain J. W. Procter and Professor J. Share Jones, the offices of the latter three gentlemen to take effect on the granting of the new Charter. It was mentioned that in making Professor Share Jones a Vice-President this was the conferring of an honour on the father of the Council who had first been elected in the year 1912 and who had been President in the year 1928-29.

The next item on the agenda was the election of a Treasurer, and it was unanimously resolved that Professor J. B. Buxton be reappointed Treasurer for the year 1949-50. A warm tribute was paid to his work as Treasurer during the past year, in which so much had been done to improve the buildings of the Royal College.



Royal College of Veterinary Surgeons—Luncheon at the Dorchester Hotel

The President, Professor Dalling, then moved from the chair that Mr. W. G. R. Oates be re-elected Secretary and Registrar, and he stated from his personal knowledge how much the Veterinary Surgeons Act and the many matters arising therefrom had depended upon his work during the past years.

The Council then proceeded to the election of committees and the principal matter was the choosing under the Veterinary Surgeons Act, 1948, of the Disciplinary Committee.

It was pointed out that under the Act the Committee must consist of one member who was a Privy Council nominee, four members who were directly elected by the veterinary profession in the United Kingdom of Great Britain and Northern Ireland, and four members chosen from the general body of the Council, whether appointed by the Privy Council, universities, veterinary colleges or elected members. The Committee was appointed as follows and the Council appointed Mr. H. W. Dawes to be the Chairman of the Committee:—

Privy Council: H. W. Dawes.

General Body of Council:—

University: R. E. Glover. Veterinary College: J. B. Buxton, A. A. Forsyth. Elected: L. Guy Anderson, G. F. Boddie, T. M. Mitchell, L. P. Pugh, H. Sumner.

It was emphasised that the Disciplinary Committee made the veterinary profession one of the foremost amongst the professions regulated by Parliament, in that it had all the powers of a Court of Law, and could hear evidence on oath and cause witnesses to attend before it under subpoena issued by the High Court.

Amongst other matters dealt with by the Council at the first meeting was the report of a special committee of the whole Council which recommended that the honour of Fellowship of the Royal College of Veterinary Surgeons by election be bestowed upon Mr. Howard Dawes, the outgoing President. This recommendation was adopted with acclamation.

Mr. Dawes, in a moving speech, expressed his thanks to all his colleagues on the outgoing Council and on the new Council for the work which had been done during the past years, and the support which had been given to him in the most difficult and trying circumstances. He expressed his personal thanks to the support which had been given to him by his wife and without which he would not have been enabled to carry the heavy burden of the Presidency for three years at a critical stage in the history of the Royal College.

After further business the first meeting of the reconstituted Council of the Royal College of Veterinary Surgeons was completed.

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Royal College of Veterinary Surgeons

Luncheon at the Dorchester Hotel

On the occasion of the first meeting of the Council of the Royal College of Veterinary Surgeons as reconstituted under the Veterinary Surgeons Act, 1948, a luncheon was held at the Dorchester Hotel, Park Lane, on June 2nd, 1949.

In the chair was the newly elected President, Professor Tom Dalling, Chief Veterinary Officer, Ministry of Agriculture and Fisheries.

As principal guests were the Rt. Hon. Tom Williams, Minister of Agriculture and Fisheries; the Rt. Hon. Viscount Addison, Lord Privy Seal and former Minister of Agriculture and Fisheries; the Rt. Hon. R. S. Hudson, M.P., former Minister of Agriculture and Fisheries; the Earl of Huntingdon, Parliamentary Under-Secretary to the Minister of Agriculture and Fisheries; Lord Lucas of Chilworth; Lord Amwell; Sir Donald Vandepeer, Permanent Secretary, Ministry of Agriculture and Fisheries; Sir Wilson Jameson, Chief Medical Officer, Ministry of Health; Sir Edward Mellanby, Medical Research Council; Dr. W. K. Slater and Mr. Havelock, Agricultural Research Council; Dr. Trueman, University Grants Committee; Mr. Harold Woolley, Deputy President of the National Farmers' Union; Sir Archibald Weigall, President, Royal Agricultural Society of England; Mr. Alec Hobson, Secretary of the Royal Agricultural Society of England; Mr. F. M. Garnham, Master of the Worshipful Company of Farriers.

Also present were representatives of the Universities of Cambridge, Glasgow, Edinburgh, London, Liverpool and Bristol; veterinary representatives of the Republic of Ireland; representatives of the Royal Army Veterinary Corps, the National Veterinary Medical Association, the Colonial Veterinary Services, and the Veterinary Services of the Commonwealth, and many other bodies.

The President, Professor Tom Dalling, stated that this was an historic occasion, and that it was very pleasing to all members of the Council to see so many persons so closely associated with veterinary science and with agriculture who had come to honour the Royal College of Veterinary Surgeons on this occasion. Although there were many representatives of agriculture present it especially gave great pleasure to see the Minister who had eventually been primarily responsible for the Veterinary Surgeons Act, 1948, and he called upon Mr. Tom Williams to say a few words.

The Minister expressed his great pleasure in being present; he mentioned that this was the first reconstitution of the Council since 1844. Although there had been differences in the past, he was glad that now the Act was implemented the recommendations of the Committee presided over by Dr. Thomas Loveday, whom everybody was glad to see present, had been given effect. He was especially glad to see Mr. Hudson who, as Minister, had been responsible for much of the initial work in the preliminary stages leading to the Act.

He knew how very much the Lord President of the Council, Mr. Herbert Morrison, regretted that he could not be present owing to other duties in as much as direct representatives of the Privy Council now sat on the Council of the Royal College.

That so representative a gathering was present was an indication of very happy relations for the future between all parties concerned. He mentioned, in particular, the role which veterinary science played in agriculture, and also the place which agriculture now occupied in the national economy of the country. It gave him great happiness that the first meeting of the reconstituted Council had taken place so harmoniously and he expressed every good wish for the future.

The immediate Past-President, Mr. Howard Dawes of West Bromwich, thanked the Minister and all the distinguished guests who were present. He recalled the long negotiations which had taken place before the implementation of the Veterinary Surgeons Act, 1948, and he placed on record the good will and amicable feeling with which the new Council had met on the previous day for the first time.

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Meat Inspection: Interdepartmental Committee to be Appointed

Prior to the rising of the House of Commons for the Whitsuntide recess, the Minister of Food announced that he had decided to appoint an interdepartmental committee to consider the present arrangements in England and Wales and in Scotland for the inspection of home-killed meat and the certification of imported meat, and to make recommendations for their improvement.

The Minister also announced his decision to appoint a working party to consider the whole question of hygiene in the trades concerned with the manufacture of meat products, and to make recommendations.

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PERSONAL

Appointment.—The appointment in the Colonial Veterinary Service is announced of Miss Margaret Massie, M.R.C.V.S., of Hereford, as Veterinary Officer, Tanganyika Territory.

Births.—Bugg.—On May 5th, 1949, at The Gables Nursing Home, Aylesbury, to Phyllis, wife of R. R. Bugg, M.R.C.V.S., a son (Richard)—brother for Patricia.

Salisbury.—On March 9th, 1949, at the Luton and Dunstable Hospital, to Isobel (*née* Murray), wife of Norman Salisbury, B.SC., M.R.C.V.S., a daughter.

Skyrme.—On May 29th, 1949, at Wokingham Regional Hospital, to Ray (*née* Rowley), wife of Harold P. Skyrme, a daughter.

Marriage.—Rogers—Smith.—The marriage took place on May 14th, 1949, between Michael J. H. Rogers, M.R.C.V.S., son of Mr. and Mrs. J. Rogers, Little Kimble, Aylesbury, and Audrey G. Smith, M.R.C.V.S., daughter of Mr. and Mrs. G. C. Smith, The Grange, Hillend, Upton-on-Severn.

Forthcoming Marriage.—Wood—Birks.—The engagement is announced between John Gerard Prescott, only son of Mr. and Mrs. E. A. P. Wood, of Tavlors, Slinfold, Sussex, and Heather Mary, younger daughter of Mr. and Mrs. C. D. Birks, of Blackfriars Farm, Horsham, Sussex.

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THE LATE MAJOR W. SHERRIFF

A Tribute

Major Sherriff was born in Dalkeith in 1863 and qualified at Edinburgh in 1890. He was in practice in Stevenage, Bath and London.

At the outbreak of war in 1914, he went on transport duty, crossing the Atlantic five times with shiploads of mules and horses. He was torpedoed in June, after which he was sent out to New Orleans from the Horse and Mule Commission. He later

returned to England and joined the R.A.V.C., and eventually became D.A.D.V.S. for the Mersey defences. Major Sherriff returned to civilian life in December, 1919, and after a short spell in practice, retired and took over a nursery in Stevenage, where he was able to enjoy to the full his favourite hobby of gardening.

One of the earliest members of Stevenage Branch of the British Legion, Major Sherriff was branch chairman for 10 years and president for a further 15 years. He was also a member of the Benevolent Fund, to which he devoted much of his time and attention from its inception.

He became a member of the Stevenage Urban Council in 1934, and for the last five of the 10 years in which he served on that body, was vice-chairman.

He was also a former chairman of Stevenage Horticultural Society, and from 1939 to 1944 was chairman of Stevenage Food Control Committee.

Major Sherriff was another of the old school of really experienced horse surgeons and he was always only too willing to help the younger members of the profession and give them the benefit of his vast experience.

He is survived by a wife and a son, Mr. A. W. Sherriff, also of Stevenage.

The funeral was on Saturday. A service at Holy Trinity Church, conducted by the rector, the Rev. J. H. King, assisted by the curate, the Rev. E. J. Harper, preceded interment in the cemetery. A guard of honour was formed by some 30 members of the British Legion and the Standard was carried.

G. K. B.

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FUNERAL OF MR. D. S. CAMPBELL, M.R.C.V.S.

The funeral of the late D. S. Campbell, M.R.C.V.S., who lost his life so tragically at Biarritz, took place in Greenock on May 27th. Members of the profession present were:—

James Smith, Ministry of Agriculture, Mold (representing the Lancashire Veterinary Association); J. G. McGregor, Ministry of Agriculture, Ministry of Food, Paisley; A. A. Hamilton, Ministry of Agriculture, Glasgow; W. D. Macrae, Ministry of Agriculture, Paisley (representing the Ministry of Agriculture).

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CENTRAL LIBRARY FOR ANIMAL DISEASES

Accessions for Quarter Ending May, 1949

PRESENTATIONS

Books

DOWNING, J.: A treatise on the disorders incident to horned cattle, etc., n.d. Presented by A. J. Baxter, Esq., M.R.C.V.S., D.V.S.M.

HANSEN, J. P., and VEJLSTRUP, C. G.: Den Danske Dyrlaegeforening, 1849-1949. Danske Dyrlaeger, 1779-1947. A/S Carl. Fr. Mortensen Kobenhavn, 1949. Presented by J. Tweedale Edwards, Esq., M.R.C.V.S.

NATIONAL VETERINARY MEDICAL ASSOCIATION. Conference on grassland and animal health. Report of proceedings, 1948. Presented by N.V.M.A.

STOCKHOLM. Collected papers from the Veterinary Institute (1948.) Presented by the Veterinary Institute, Stockholm.

Reports, 9; *Pamphlets and Reprints*, 71

PURCHASES

BUXTON, P. A.: Trypanosomiasis in Eastern Africa. (London, 1947.) 3s.

DAVEY, T. H.: Trypanosomiasis in British West Africa. (London, 1948.) 2s.

MERCHANT, I. A.: Veterinary bacteriology. 3rd edition. (Ames, 1946.) 42s.

NASH, T. A. M.: Tsetse flies in British West Africa. (London, 1948.) 30s.

OPPERMAN, T.: Lehrbuch der Krankheiten des Schafes. (Hannover, 1946.) 48s.

REINHARDT, R.: Lehrbuch der Geflugelkrankheiten. (Hannover, 1946.) 52s.

WAKSMAN, S. A.: The Literature on Streptomycin. (Rutgers U.P., N. Jersey, 1948.) 18s.

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POLYARTHRITIS IN LAMBS FOLLOWING SERUM INOCULATION

We have been asked by Messrs. Boots, the International Serum Co., and Bayer Products Ltd., to intimate the fact that the serum used in the outbreak of polyarthritis in lambs occurring in North Wales, to which reference was made in our issue of February 26th, was not prepared or sold by them.

R.A.V.C. GOLFING SOCIETY

Captain G. S. Walker won the Challenge Cup at the Meeting of the Royal Army Veterinary Corps Golfing Society, held on the course of the North Hants Golf Club, at Fleet, on May 27th, 1949.

Other prize winners: Founders Cup and Prize, Lt.-Col. J. Bell; Conder Cup and Prize (Eclectic), Capt. G. S. Walker; Captain's Prize, Lt.-Col. J. Bell; Club Prize, Lt.-Col. D. F. G. Smith.

The meeting was held in fine but blustery weather, and some good scores were recorded.

The following office-bearers were elected for 1949-50: President, Col. A. G. Todd, C.B.E., D.S.O.; Vice-president, Col. W. H Walker, O.B.E., D.S.O.; Captain, Lt.-Col. J. Bell; Committee, Col. R. M. Bamford, O.B.E., Lt.-Col. R. H. Knowles, Capt. G. S. Walker; Hon. Secretary and Treasurer, Capt. J. H. Wilkins, R.A.V.C.

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Use of L-Thyroxine by Mouth for Stimulating Milk Secretion in Lactating Cows

The following letter on the above subject is contributed to the issue of *Nature* of May 21st by G. L. Bailey, S. Bartlett and S. J. Folley, of the National Institute for Research in Dairying:

Since it was first unequivocally shown that the milk yield of lactating cows could be increased by feeding dried thyroid gland[1] or injecting thyroxine[2,3], many experiments (see Reineke[4] for review of some of them) have been carried out on the practical use of thyroid-active substances for increasing the milk yield of cows over limited periods of time. Dried thyroid gland and, more recently, iodo-casein (an artificially prepared thyroid-active protein) have been preferred to synthetic thyroxine for this purpose, because of the high cost of the latter and the belief that its activity was not great by the oral route.

In any event, the galactopoietic effects of thyroxine administered to cows by mouth have not yet, so far as we know, been investigated. However, the recent development of new methods for the synthesis, first of racemic thyroxine[5], and later of L-thyroxine[6], which holds out considerable promise that thyroxine will eventually be made available on a large scale at a cost low enough to compete with iodo-casein, has now prompted an investigation of the effects of thyroxine, given by mouth, on the milk yield of cows in declining lactation.

Dose/cow/day	Milk yield increase (lb./cow/day)		Heart-rate increase (beats/cow/min.)	
	after 2 weeks	after 3 weeks	after 2 weeks	after 3 weeks
25 mgm. L-thyroxine	1·5	1·6	1·8	5·2
50 mgm. „	3·2	3·6	6·2	8·7
100 mgm. „	6·2	5·9	13·5	15·5
150 mgm. „	6·3	6·3	16·9	21·5
15 gm. iodo-casein	4·9	4·3	7·4	11·2

Forty-eight cows in equal numbers on two farms, comprising eight blocks each of six matched cows, that is, six treatment groups, were used in an experiment involving the feeding of L-thyroxine (as sodium salt) at four dose-levels, iodo-casein at one, and a control group. The thyroxine, incorporated with oatmeal and made into tablets each containing 25 mgm., was fed twice daily for 21 days. The responses given in the accompanying table were calculated from the means of readings for the 12th, 13th and 14th days, and the 19th, 20th and 21st days respectively. In the case of milk yield, the figures for the three days immediately before treatment started were used as a standard of comparison, the responses being calculated by the formula: (milk yield of treated cows in treatment period—milk yield of control cows in treatment period)—(milk yield of treated cows in control period—milk yield of control cows in control period).

It will be seen that at the three lower dose-levels of thyroxine graded responses were obtained both with milk yield and heart-rate; but 150 mgm. appeared to be an overdose, as evidenced by slight sweating of a few cows after 14 days. The 15 gm. of iodo-casein, which was chosen as the optimum dose of that preparation, was estimated by linear interpolation to be equivalent to 60 to 78 mgm. of L-thyroxine. Since iodo-casein is believed to contain about 3 per cent. L-thyroxine[4], this comparison raises the alternative possibilities that these published estimates of the thyroxine content of iodo-casein preparations are in error, or that the efficiency of oral utilisation of thyroxine combined in iodo-casein is much lower than that of the pure hormone.

In a previous experiment involving 14 daily subcutaneous injections of 10 mgm. DL-thyroxine into lactating cows, Folley and

White[3] obtained a response at the end of the second week of the injection period amounting to approximately 5 lb. milk/cow/day. From the data in the table, linear interpolation shows that a response of about this magnitude was given by approximately 82 mgm. L-thyroxine administered by mouth. Taking the relative biological activities of L- and DL-thyroxine as very near 2 : 1[4,7], it follows that the oral/parenteral ratio for L-thyroxine in the cow is approximately 16 : 1.

The effects of the treatments on the fat and non-fatty solids (no significant changes), lactose, protein and nitrogen distribution of the milk have been studied by Dr. S. J. Rowland; on certain milk vitamins by Dr. S. Y. Thompson; and on milk iodine by Dr. H. H. Green. These results, together with the changes in milk phosphatase, will be fully reported later.

Since these results seem to open up the possibility of using synthetic L-thyroxine in practice for stimulating milk yield in cattle, an examination of the advantages of the pure hormone over iodo-casein may be of interest. These are: (*a*) there is no necessity for bioassay, a troublesome and unreliable procedure unavoidable with iodo-casein; (*b*) the total iodine fed is much less than with a dose of iodo-casein of equal activity and there is no risk of iodism; and (*c*) L-thyroxine seems to be virtually tasteless and when incorporated into suitable tablets is readily eaten by cows, which is not always the case with iodo-casein.

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PHYSIOLOGICAL STRAIN OF HIGH MILK YIELDS

With the greater physiological strain now being imposed on our dairy animals by the extraction of high yields of milk, is there not a great need for research into the effect on the animals' constitutions? This question, by Dr. H. D. Kay, was one of many which were asked at the end of a talk on the future farm possibilities of disease-free herds, given by Mr. J. C. Buxton at a meeting of the Society of Dairy Technology in London last week (states *The Farmer and Stock-Breeder* in the report of the proceedings appearing in its issue of May 17th).

Mr. Buxton, who is veterinary investigation officer at the Nottingham University School of Agriculture, Sutton Bonington, replied: "We have always got to remember that the cow is an animal and not a machine for turning feed into milk." Describing high yields as an exploitation of the physiology of a cow, he emphasised that any additional strains to be placed on the animal in raising yields should follow in the wake of the increase in our knowledge of her physiology, rather than precede it.

It is probably safe to predict that the era of the protection of calves from an early age against tuberculosis is at hand, said Mr. Buxton in the course of his address. Discussing the possibilities of control in the future Mr. Buxton said that vaccination of stock by the use of B.C.G. may be practicable in a short time. Although not harmful, the vaccine is alive and has the disadvantage that it leaves in its wake a positive reaction to the tuberculin test, so that following vaccination it is not possible to detect the presence of an active field infection in the body in the event of immunity waning or breaking down. There is experimental evidence to support the belief that a lifelong immunity can be conferred by the administration of B.C.G. vaccine and that the danger of its setting up active disease is extremely remote. "There is no doubt that it is possible, by the application of diligence and the use of a reliable diagnostic agent, to remove from a herd which is under suitable management the danger of infection with tuberculosis," he said. "However, in addition to the disposal of reactor cows it is important that the hygiene and herd management should be of a consistently high standard in order that the general level of health in the herd shall be maintained at its best.

"In this respect it is worth while to devote attention to drainage and ventilation in the cow byre and wherever possible it is better that rows of cows should face away from each other. But in a self-contained and T.T. herd it is doubtful whether there would be sufficient fresh infection between periodical tests to create a general danger to the healthy members of the herd.

"In the so-called 'flying herd,' however, in which the population is often changing, when fresh cows are being added frequently from unknown sources, the whole problem of the maintenance and the health of the community becomes complex, and it is probable that such herds act as disseminators of disease and particularly of tuberculosis on a considerable scale. Under such conditions of management it is often impossible to avoid at times the importation of the actively open case of tuberculosis."

Mr. James Mackintosh, who opened the discussion, referred to the difficulty caused by the positive effect of an S.19 vaccination on a succeeding agglutination test, and asked that a national scheme of calf identification should be started. He also spoke of the need for research into "what goes by the omnibus name of sterility, which is causing great concern."

On a point made by Mr. E. Rae that farmers were still prejudiced against the mixing of poultry with an attested herd for fear of tuberculous infection, Mr. Buxton said he thought there was always a danger that avian tuberculosis might affect a bovine tuberculin test.

Dr. R. L. Provan thought that publicity should be given to the fact that the eradication of herd diseases was not such a difficult task as it was often made out to be.

Mr. Ben Davies doubted whether there had been any overall reduction in the number of tuberculous dairy animals in the country during the past 30 years. "We all know what happens to reactors: they go to market and then on to produce milk for somebody with an unattested herd."

Dr. Kay asked that more instruction in the value of cowmanship in reducing the incidence of disease should be given by the N.A.A.S.

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MINISTRY OF AGRICULTURE NEWS SERVICE

Up-Grading the Flying Herd

To-day a large proportion of the herds in Lancashire and the industrial parts of the West Riding fall under the heading of "flying herds," that is, herds where the owner does not breed his own dairy replacements but is dependent on brought-in animals. Systems of management such as the disposal of cows from the herd and the intake of fresh animals are governed largely by local conditions and custom, but the old "milk and feed" system has, with a few exceptions, practically disappeared. In many of the larger flying herds a bull is maintained, but it is fair comment to say that, generally speaking, the bulls used in these herds are of a lower standard than those kept in the self-contained herds.

On most farms with flying herds, few of the premises are suitable for calf rearing, and calves are disposed of soon after birth to the collecting centres. Improvement schemes and methods of grading-up introduced in the past have not always been capable of practical application to flying herds, but this side of the milk-producing industry has a definite part to play in the expansion programme, and the best use must be made of this reservoir. The great problem which faces these herd owners year after year is the question of replacements, and for many it is a complete gamble from both the health and the production angles. What is wanted is a system of management which will overcome this unsatisfactory state of affairs. Fortunately, there is an answer—artificial insemination.

With A.I. Centres and their sub-stations extending over the country, flying herd owners have a remedy and with it a great opportunity to help themselves and the expansion programme. The bulls at these centres have been selected with great care, particular attention being paid to their breeding, milk and butterfat ancestry, and conformation. Cows which are suitable for breeding dairy replacements should be inseminated with semen from a bull of the same breed. Low-yielding cows suitable for breeding beef stores should be inseminated with semen from colour-marking bulls under the free service beef scheme.

So far so good: but steps must be taken to ensure that the resulting progeny do not go to the collecting centre for slaughter. These calves should be sent either direct or through the store markets to healthy rearing farms, the heifers out of suitable dams being reared on for dairy replacements, and the steers, where suitable, and all colour-marked calves, should in due course meet a ready demand as beef stores. The calf rearer seeking sources of supply would thus be provided with a selection of suitably bred calves, and if arrangements were made for the identification (by ear-marking) of the heifer calves it is felt that the demand by flying herd owners to get these well-bred heifers back as replacements would well repay the rearer. The milk producer securing these replacements should reap a rich dividend and largely eliminate the element of chance associated with the present system of buying in replacements.

Feeding-stuffs Rationing Offences

A farmer was recently fined £80 with costs for offences against the Feeding-stuffs (Rationing) Order. When claiming rations for his dairy cows for November last, the farmer (who is a retailer as well as a producer) stated that his September milk sales had been 4,636 gallons, and rations were accordingly issued on this figure pending the usual verification from his M.M.B. Account form; it was later discovered from this form that the actual sales figure for September was only 2,065 gallons. Similarly, the farmer claimed December rations based on 4,651 gallons of milk sold in October, whereas the true figure was only 2,131 gallons.

In imposing the fine the Chairman of the Bench pointed out to the defendant that by obtaining more feeding-stuffs than he was entitled to he was depriving other farmers of their fair share.

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CRUELTY TO WILD ANIMALS COMMITTEE

Members will be interested to learn that it was announced in the House of Commons on Thursday of last week that Major L. P. Pugh, B.Sc., F.R.C.V.S., of Sevenoaks, has been appointed a member of the above Committee. The other members are: Mr. Charles W. Brandon, J.P., Mr. W. J. Brown, Mr. John Cripps, Professor P. B. Medawar, M.A., D.Sc., Mr. H. Burn Murdoch, LL.D., Miss Frances Pitt, J.P. Mr. John Scott Henderson, K.C., will preside.

In reply to a request for information regarding the qualifications of the members, the Home Secretary stated: "Mr. Brandon is area secretary of the Transport and General Workers' Union—(laughter)—and is especially qualified to speak on the views of rural workers. Mr. Brown is a barrister—(laughter)—and a well-known contributor to the *Field*. Mr. Cripps is editor of the *Countryman*. Professor Medawar is professor of zoology at Birmingham. Mr. Burn Murdoch was lately Sheriff Substitute at Sterling. Miss Pitt is a naturalist, and writes a popular feature article in the *Evening News*, an article which sometimes well expresses the normal person's attitude to this kind of thing. Major Pugh is a veterinary surgeon."

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OFFICIAL OPENING OF NEW JOINT FACTORY

The formal opening of the new joint factory of Newton Victor, Ltd., and Metropolitan-Vickers Electrical Co., Ltd., at Motherwell, Scotland, took place on May 24. Newton Victor production in the new works is concerned with medical and industrial X-ray equipment and physiotherapy apparatus and, on the Metrovick side, with electrical meters and instruments.

Some 70 guests were received at the works by Mr. Oliver Lyttelton, D.S.O., M.C., M.P., Chairman of Associated Electrical Industries, Ltd., and conducted on an inspection tour of its various departments. Later they attended a dinner at the Central Hotel, Glasgow, to mark the official opening, at which Mr. Oliver Lyttelton presided, and at which the principal speaker was the Rt. Hon. Tom Johnston, P.C., Chairman of the Scottish Hydro-Electric Board and former Secretary of State for Scotland.

On the Newton Victor side of the Motherwell works activity is wholly devoted to the production of X-ray equipment for medical, dental, veterinary and industrial purposes.

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The Duke of Wellington told the annual meeting of the Council for Preservation of Rural England in London that he would like to see it as a Council for the Preservation of Agricultural England and not so much a Council for the embalming of Shakespeare's England.

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To prevent spurious pedigrees being submitted claiming famous dogs as sires to litters, the International Sheep Dog Society now requires owners of dogs to forward notice of every service on a postage paid printed form supplied by the Keeper of the Stud.

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the N.V.M.A.

Correspondents are requested to write as briefly as possible.

PERSONNEL OF THE PROFESSION

Sir,—My remarks, to which Miss Ford objects, amounted to the logical conclusions of a series of thoughts arising partly from observance of the working of the National Health Service. Miss Ford insisted that all disease ought to be treated. Good, so the first requirement to this end is the deprival of all animal owners of their power of decision about sickness in their livestock and the vesting of this decision in "hordes of officials" (veterinary and ancillary), all answerable to one chief; hence my "totalitarian."

The remuneration of this horde would call for an increase in taxation of one form or another, just as is happening in the N.H.S.; hence my remark about standard of living. It is not the fact, as the Socialists make out, that what people cannot afford individually they can afford collectively!

Incidentally, animal owners treated in the way referred to above become mere animal lodging place keepers, forced to obey orders in the management of their stock—another aspect of totalitarianism.

About Mr. Hughes's plea. While commending it, one finds little encouragement from history.

Yours faithfully,

37, Hawthorn Lane. J. Tweedale Edwards.
Wilmslow, Cheshire.

May 29th, 1949.

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Sir,—In my letter under the heading of "The Future of the Profession," through an oversight, I omitted to give an answer to the six questions put by my friend L. Guy Anderson, in his communication which appeared in the issue of April 30th.

My opinion is, that the answer to the first five is in the negative: the sixth, affirmative.

Yours faithfully,

"Rothiemurchus," J. F. D. Tutt.
St. Cross, Winchester.

May 23rd, 1949.

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A ROYAL COLLEGE OF VETERINARY SURGEONS COUNCIL RESIGNATION

Sir,—Mr. Arnold Spicer's letter in your issue of May 28th, 1949, calls for comment.

The last few lines in the pamphlet issued under the authority of the R.C.V.S. Council states that every case will be dealt with on its merits. It could not very well be otherwise. Unless Mr. Spicer has a different view from mine as to what constitutes a penalty, I do not understand his statement that the case in question "was dismissed without a penalty of any sort."

Mr. Spicer says "If the profession knew the whole facts of the case, it is quite certain that, whilst questioning the wisdom of Mr. Steele-Bodger's action in resigning from the Council, they would fully agree with his reasons for taking that step." I doubt if anyone is qualified to express a dogmatic opinion as to what the profession would agree with before it had access to the facts.

Decisions as to the nature of the punishment to be inflicted on offenders are controlled by regulations of the College. To prevent a miscarriage of justice in some circumstances, a stipulated majority of votes controls the issue. If that majority does not exist then the course of action has to be adjusted accordingly.

In his final paragraph, Mr. Spicer says "The trouble is that some members of Council have so much sympathy for the culprits that they have none left for the probable victims of these gross breaches of the Ethical Code."

This again is a dogmatic statement. Surely Mr. Spicer realises that there is at least one other reason which influences those who do not think just in the way he expresses himself. The degree of punishment which might be ignored by one person might very easily do another far more damage than was intended, or was necessary for the common good.

Yours faithfully,

Penzance. S. J. Motton.

May 31st, 1949.